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International Journal of Orthodontia and Oral Surgery

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International
Journal of Orthodontia
and Oral Surgery

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VOL. 22 MAY, 1936 No. 5

Orthodontia

ROOT RESORPTIONS AND THEIR RELATION TO PATHOLOGIC BONE FORMATION*

PART I: STATISTICAL DATA AND ROENTGENOGRAPHIC ASPECT†

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SAN FRANCISCO, CALIF.

ALMOST a decade has elapsed since Ketcham brought to the attention of the profession his findings regarding the resorption of the roots of teeth following orthodontic treatment. These findings, amply supported by definite roentgenologic evidence, were received with considerable astonishment by the orthodontic practitioner. What was the reason for the high percentage (21 per cent of the cases examined) of root resorptions observed in those treated cases? Was it the type of appliance used? Was it the degree of force applied? Did it have any relationship to the orthodontic treatment, or was it the usual result of some hidden factors not connected with orthodontic treatment at all? For nine years we have been searching for the answers to these questions. During this time our efforts have been aimed at determining the true etiology and histopathogenesis of these cases of root resorption of which there has been as yet only roentgenologic evidence.

It is still impossible to say in any given case of root resorption just what is the specific factor contributing to this condition. As yet no one factor *alone* and by itself can be made responsible for those resorptive processes not connected with periapical infections or extreme mechanical stress or pressure. It is impossible to say in the light of our present knowledge of the subject whether the forces involved in orthodontic treatment can be held solely to blame for the resorption of roots so frequently observed in treated cases.

*Conducted under grants made by the California State Dental Association, California Academy of Periodontology, and the Research Board of the University of California.

Presented before the Thirty-Third Annual Meeting of the American Society of Orthodontists in New York City, May 1, 1935.

†Part II containing more detailed records of the individual cases will appear at a later date.

The orthodontic practitioner up to the present time has observed these changes and, in silence, has pondered over his responsibility regarding them. Time and again he has filed the seemingly damaging roentgenologic evidence in his cabinet and waited for some explanation of this phenomenon which would relieve his conscience of its sense of guilt. The desire to be able to foretell in which cases he could logically expect these resorptive processes to take place and in which cases he could be assured they would not occur has become increasingly acute in the mind of the practitioner.

From the *histopathologic* point of view we have gained considerable knowledge during the last few years concerning changes in the paradentium which occur in the course of orthodontic treatment in man (Herzberg), and in experimental animals (Gottlieb, Johnson, Appleton and Rittershofer, Kronfeld, Marshall, Oppenheim and others). We know now that we must not exceed the capillary blood pressure in the peridental membrane during orthodontic movement of teeth in order to promote uniform bone resorption and new formation of alveolar structure without injuring the tooth surface (Schwarz).

Excellent reviews of the work of other authors who have contributed to this most important subject for orthodontic practice make it unnecessary here to summarize again what has been done in this field so far.

From a *practical* point of view it seems as though the question of determining factors which influence the individual susceptibility to root resorption is the most important one, because the nature of these factors determines to a large extent the frequency of these lesions. It is the general experience that in certain patients an excessive orthodontic force will not cause any changes of the root surface while moderate force in others will produce tremendous root resorption! Observations have been reported which indicate that root resorptions can occur *even without* orthodontic treatment (Chipps, Gottlieb, Ketcham, Kronfeld, Müller-Rony, and others). This latter type of root resorption is designated as idiopathic root resorption, because it occurs as the result of unknown causes.

We are well informed about the etiology of other large groups of root resorptions. We know that *inflammatory processes* within the paradentium can lead very rapidly to resorptions of hard substances as, for instance, paradental or periapical abscesses, acute or chronic forms of periodontitis, etc. (Figs. 1-4.)

We also know that pressure can lead to similar changes in the form of *pressure atrophy.*

Teeth in close proximity to tumors and cysts (Figs. 5-7) are frequently subject to such a pressure atrophy, while the presence of impacted teeth often results in the resorption of the roots of those teeth adjacent (Fig. 8).

The resorptions of replanted teeth must be grouped separately because of tissue changes in the paradentium resulting from chronic irritation by the replanted tooth.

Of all the forms of root resorption with which we are familiar, the one form with which the dental profession, and in particular the orthodontist, is most concerned is the idiopathic form. What he wants to know is: Does the idiopathic form of resorption constitute a certain proportion of the cases he has

observed following orthodontic treatment? Is it possible that the unknown factors in the etiology of the idiopathic form are responsible for some of the root resorptions heretofore explained solely on the basis of mechanical influences?

Very few attempts have been made to determine the biologic factors involved in the so-called idiopathic resorptions or the resorptions following orthodontic treatment. Even fewer attempts, if any, have been made to determine whether or not both forms may have identical biologic backgrounds. In most in-

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Figs. 1, 2, and 3.—Apical root resorptions due to chronic inflammatory processes.
Fig. 4.—Apical root resorption following filling of root canal.

stances the resorptive process has been explained as being due to mechanical stress alone, without studying in detail the systemic condition of the patient which might have brought about an individual susceptibility to increased resorptive activity of bone and teeth.

The results of recent animal experimentation called to our attention the fact that root resorptions can occur on the basis of dietary deficiencies (Becks-Weber).

It was found that diets with *normal salt mixtures,* as well as those with *low calcium salt mixtures,* both without the addition of cod liver oil, given to a

series of dogs, led to atrophic and dystrophic bone changes of the entire skeleton, including the jaws (Figs. 10 and 11).*

These more or less severe bone disturbances were accompanied by marked resorptions of the roots of the teeth in these animals.

Fig. 5.

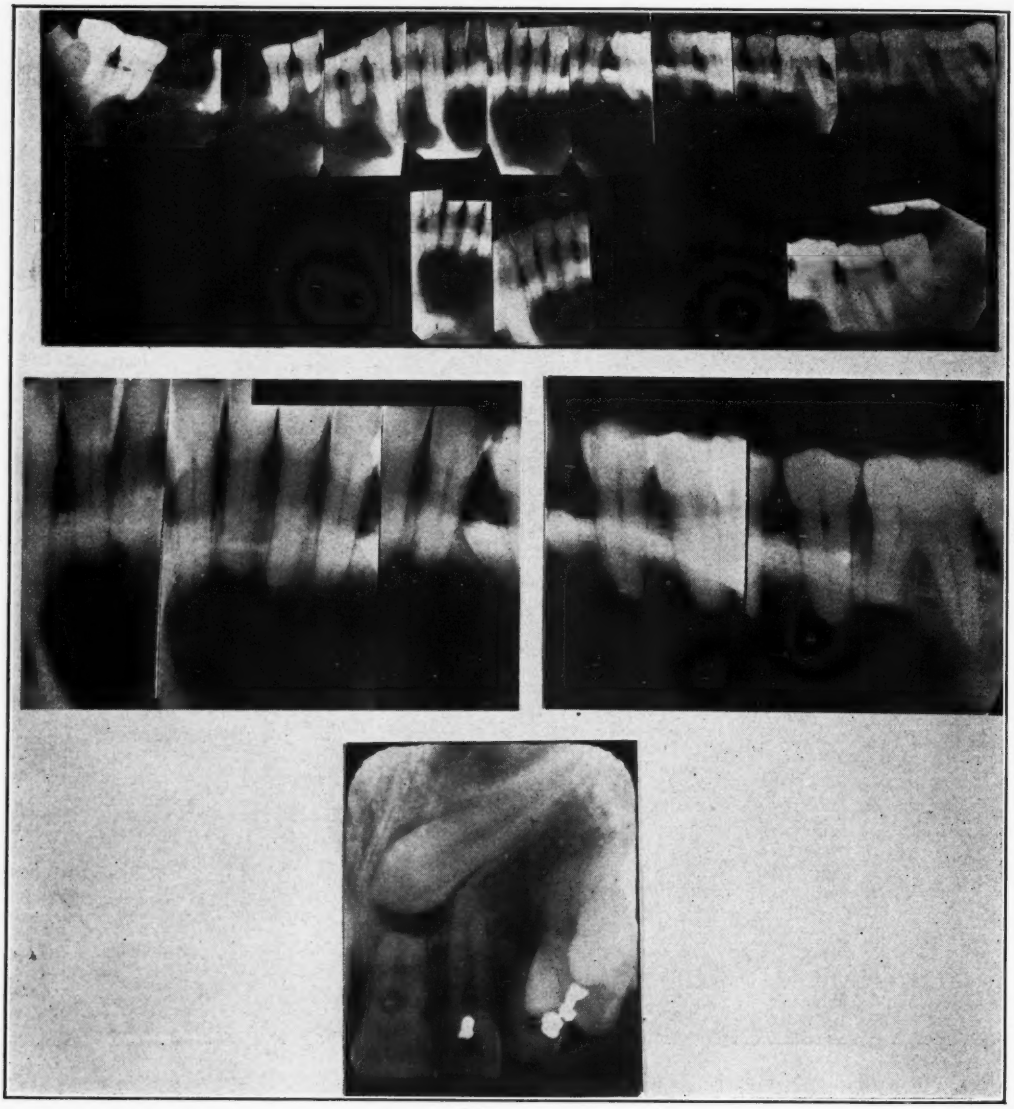

Fig. 6. Fig. 7.

Fig. 8.

Fig. 5.—Large follicular cyst extending from first left mandibular molar over entire anterior region to the apices of the mandibular right second molar. Cyst originated from supernumerary tooth under apex of right mandibular cuspid. Multiple root resorptions in the involved area. Patient aged sixty-five years.

Fig. 6.—Enlargement of anterior region in Fig. 5.

Fig. 7.—Enlargement of mandibular left bicuspid and molar areas in Fig. 5.

Fig. 8.—Root resorptions of central and lateral incisors due to eruption of cuspid (pressure atrophy).

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*Figs. 9, 10 and 11 are taken from: Becks, Hermann, and Simmonds, Nina: Dental Caries and Paradental Disturbances: I. Importance of an Adequate Diet for Health of Teeth and Paradentium, J. A. D. A. 22: October, 1935.

Since these resorptions of roots occurred in varying degrees on practically every tooth of these animals, the conclusion was drawn that the local tissue reaction, together with the tremendous increase in resorptive activity, which is characteristic for this type of osteoporosis and also for the osteodystrophic lesions, was also responsible for the loss of apical tooth structure. In other words, the root resorption in these experimental animals was the expression of a generalized disturbance caused by a primary nutritional deficiency.

Fig. 9. Fig. 10.

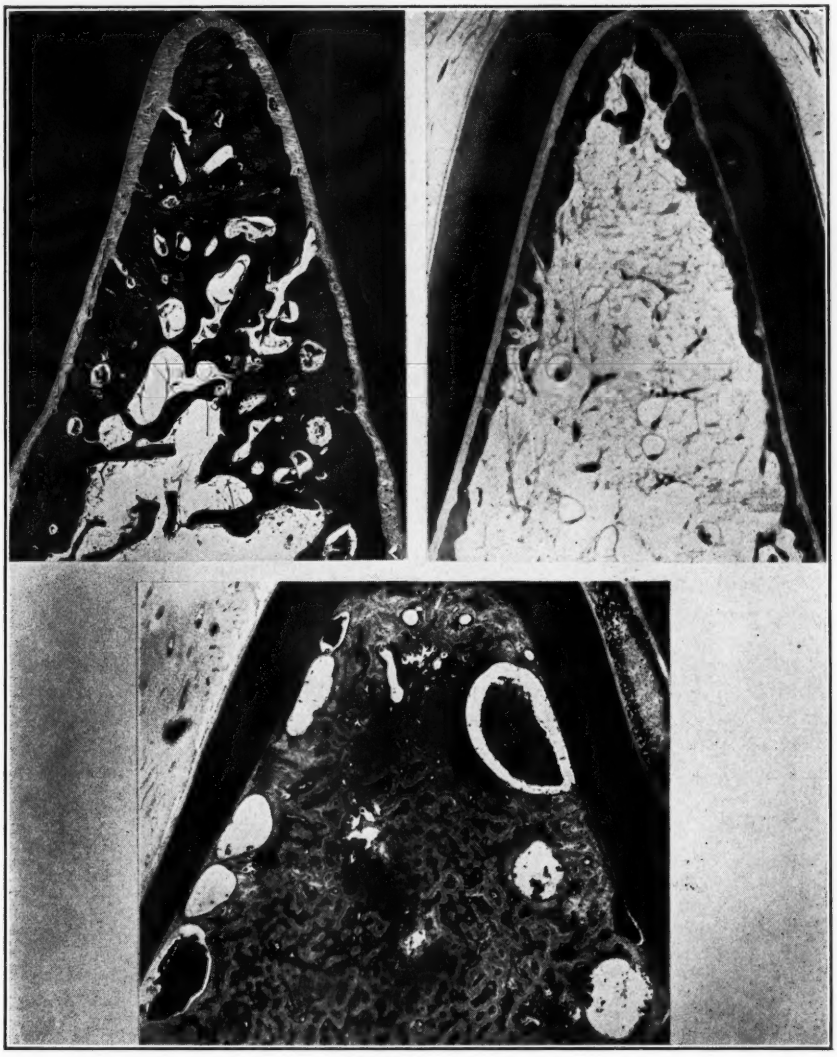

Fig. 11.

Fig. 9.—Normal bone formation under bifurcation of premolar of dog. Marrow cavities appear small; bone deposition and physiologic resorptive process seem to keep balance. (Specimen No. 998A.) (Microsummar, 42; extension of camera, 33 inches; distance from object, 4 cm.)

Fig. 10.—Severe osteoporosis (progressive bone atrophy, Askanazy) under bifurcation of a premolar. Dog received a normal salt mixture diet without the addition of cod liver oil. Most of the bone trabeculae (supporting bone) have been resorbed, leaving only the alveolar bone. Blood vessels are tremendously enlarged. (Specimen No. 2056.) (Microsummar, 24; extension of camera, 20 inches; distance from object, 1.5 cm.)

Fig. 11.—Osteodystrophia fibrosa with cystic developments under the bifurcation of a premolar. Dog received a low calcium salt mixture without the addition of cod liver oil. The original bone structure has been completely resorbed and is replaced by a deficient dystrophic type of bone. (Specimen No. 1795.) (Microsummar, 42; extension of camera, 31 inches; distance from object, 1½ inches.)

In recent animal experiments Jaffe and Bodansky have given a daily dose of two units of parathormone per kilo body weight which was gradually raised in some dogs to as high as five units toward the end of the experimental period of five to six months. This produced all degrees of change from mild bone resorption and a slight fibrous replacement of the marrow to severe resorption

Fig. 12. Fig. 13.

Fig. 14.

Fig. 12.—Multiple resorptions of surface of root (dog) with dystrophic bone lesion as the result of a low calcium salt mixture diet and without the addition of cod liver oil. (Specimen No. 2044 B.) (Leitz apochromatic, 16 mm.; eyepiece 4 ×; extension of camera, 52.5 cm.)

Fig. 13.—Root resorption accompanying osteodystrophia fibrosa. (Specimen No. 1778.) (Leitz objective, 1 b; eyepiece 4 ×; extension of camera, 77.5 cm.)

Fig. 14.—Advanced resorption of root end of premolar of a dog suffering from osteodystrophia fibrosa as the result of a low calcium salt mixture diet without the addition of cod liver oil. Newly formed bone structure consists of soft, deficiently calcified, fibrous osteoid tissue. (Specimen No. 2027.) (Leitz objective, 1 b; eyepiece 4 ×; extension of camera, 82.5 cm.)

and degeneration of the marrow with hemorrhage when the animal died of overdosage. Clinical observations have confirmed, to some extent, the finding that osteodystrophic changes of the skeleton are frequently the result of parathyroid tumors. Jaws and teeth of these animals apparently were not examined histologically.

Reports concerning the incidence of severe osteoporotic lesions as the result of dietary deficiencies, which were observed especially during and after the World War in Europe, indicate definitely that a lack of certain food constituents can produce severe changes in the bony system in man ("Hungerosteopathy").

On the basis of such an apparently complex etiology of pathologic bone formation—and many other etiologic relations could be included—and also in view of the experiments mentioned at the beginning, it was natural to suspect that the factors related to such bone disturbances as osteoporosis and osteodystrophia fibrosa in man might also be essential factors in the production of root resorption.

Ketcham and others had already suggested that perhaps some of the observed root resorptions were the result of a nutritional deficiency without, however, having offered any definite proof of such a hypothetical contention. Since these unexpected root resorptions in animals reminded one of multiple root resorptions which Gottlieb described as typical for his so-called "diffuse atrophy," it was decided to follow this experiment by an extensive clinical investigation of patients suffering from root resorptions.

Since the mechanical factor did not seem to be the only cause for all resorptions, every possible etiologic relationship was checked on an equal basis, and the patients were submitted to the following routine procedure:

1. Complete physical examination, performed by a consulting staff and amplified by specialists in the various fields of medicine.
2. Urinalysis and differential blood count.
3. A complete dietary survey made by Dr. Nina Simmonds.
4. Clinical and roentgenographic examinations of teeth and jaws.
5. Determination of the basal metabolic rate.
6. Calcium and phosphorus analysis of blood serum and saliva.
7. Determination of bone age in patients under fifteen years of age.
8. Gnathostatic impressions of maxilla and mandible.

If the patient had been treated orthodontically, the attempt was made to secure casts and roentgenograms from the orthodontist with data as to:

1. The age of patient at the beginning of orthodontic treatment.
2. The general course of treatment.
3. Type of appliances used.
4. Exact orthodontic diagnosis of the condition at the beginning of treatment.
5. Possible pain and severe loosening during tooth movement.
6. Duration of treatment.

The most extreme cases of root resorption which were observed at the Orthodontic Department of the University of California were selected first for this analysis in order to find a lead to any possible systemic factor.

It is always well to remember that while a low calcium and low vitamin D diet may produce multiple root resorptions in animals, the results in man may not be the same. On the other hand, however, it was suspected that any other factor influencing the mineral or general metabolism might have a bearing on the problem of root resorption.

It was surprising to learn that the first six patients who submitted to this examination were pronounced by the physician as being typical *hypothyroid* cases with a basal metabolic rate below minus 20 per cent. The observations of the next few months brought to light a rapidly increasing number of patients with root resorptions. The more attention was paid to these changes, which of course could be observed only roentgenographically, the more patients were found with severe resorptions who had had *no* orthodontic treatment during childhood. Statistical data were accumulated and Table I gives a survey of the types of systemic diseases and also the frequency—expressed in percentages—in 35 treated cases and 19 cases without previous treatment.*

TABLE I

FREQUENCY OF VARIOUS SYSTEMIC DISTURBANCES IN 54 PATIENTS WITH ROOT RESORPTIONS (SERIES I)

		35 PATIENTS WITH PREVIOUS ORTHODONTIC TREATMENT		19 PATIENTS WITHOUT PREVIOUS ORTHODONTIC TREATMENT	
		NUMBER OF CASES	PERCENTAGE	NUMBER OF CASES	PERCENTAGE
I	Hypothyroidism	21	60	9	47.3
II	Hypothyroidism plus other endocrine disturbances	8	23	6	31.5
	Total of I and II	29	83	15	78.8
III	Endocrine disturbances without hypothyroidism	4	11.3	2	10.6
	Total of I, II, and III	33	94.3	17	89.4
IV	Systemic disturbances other than endocrine unbalance	2	5.7	2	10.6

It may be noted that the total frequency of endocrine disturbances in patients with previous orthodontic treatment was as high as 94.3 per cent, which was only very slightly different (less than 5 per cent) from the untreated group (89.4 per cent). This difference was explained as due mainly to the smaller number of patients *without* orthodontic treatment examined up to that time. The investigation was, therefore, continued, and special attention was given to cases which had never been cared for by an orthodontist.

Our material has now reached a number which seems to justify a comparison of the two groups, and Tables II to V demonstrate the clinical and laboratory findings in one hundred patients of which only fifty had been treated orthodontically.

*Reported before the Twelfth General Meeting of the International Association for Dental Research in Chicago, March, 1934. (Becks, Hermann: Some Relevant Conditions of Root-End Resorption, J. Dent. Res. 14: June, 1934.

TABLE II

FREQUENCY OF ENDOCRINE DISTURBANCES AND OTHER SYSTEMIC DISEASES FOUND IN COEXISTENCE WITH ROOT RESORPTION IN 100 CASES

		50 PATIENTS WITH PREVIOUS ORTHODONTIC TREATMENT					50 PATIENTS WITHOUT PREVIOUS ORTHODONTIC TREATMENT				
		NUMBER OF CASES					NUMBER OF CASES				
		SERIES I*	SERIES II*	TOTAL		PERCENTAGE	SERIES I*	SERIES II	TOTAL		PERCENTAGE
I	Hypothyroidism	21	9	30		60	9	11	20		40
II	Hypothyroidism plus hypopituitarism	1	–	1	10	20	1	–	1	13	26
	Hypothyroidism plus hypogonadism	7	1	8			5	4	9		
	Hypothyroidism plus hypogonadism and hypopituitarism	–	–	–			–	3	3		
	Hypothyroidism plus hyperparathyroidism	1	–	1			–	–	–		
III	Hyperthyroidism	1	–	1	6	12	1	1	2	7	14
	Hyperthyroidism plus hypogonadism and hypopituitarism	–	1	1			–	–	–		
	Hyperpituitarism	1	–	1			1	3	4		
	Hypopituitarism	1	–	1			–	–	–		
	Hypopituitarism plus hypogonadism	1	–	1			–	–	–		
	Hypogonadism	–	1	1			–	1	1		
IV	Other diseases	1	–	1		2	2	5	7		7
V	Negative physical examination	–	3	3		6	–	3	3		6
	Total	35	15	50		100	19	31	50		100

*The patients listed under Series I (35 with and 19 without orthodontic treatment) were included in the first report (J. Dent. Res. **14**: June, 1934). Series II has been added to bring each group to 50, for comparative purposes.

TABLE III

TYPE AND PERCENTAGE OF SYSTEMIC DISEASES IN 100 CASES OF ROOT RESORPTION

		50 PATIENTS WITH PREVIOUS ORTHODONTIC TREATMENT		50 PATIENTS WITHOUT PREVIOUS ORTHODONTIC TREATMENT	
		NUMBER OF CASES	PERCENTAGE	NUMBER OF CASES	PERCENTAGE
I	Hypothyroidism	30	60	20	40
II	Hypothyroidism plus other endocrine disturbances	10	20	13	26
	Total of I and II	40	80	33	66
III	Endocrine disturbances without hypothyroidism	6	12	7	14
	Total of I, II, and III	46	92	40	80
IV	Systemic disturbances other than endocrine unbalance	1	2	7	14
	Total of I, II, III and IV	47	94	47	94
V	Negative physical examination	3	6	3	6

Table II demonstrates the frequency of endocrine disturbances and other systemic diseases which were found *in coexistence* with root resorption. Of all endocrine disturbances, hypothyroidism (I) shows the highest frequency, amounting to 60 per cent in the group having previous orthodontic treatment. This is 20 per cent higher than in the untreated group. The group without orthodontic treatment, however, shows a higher frequency of pluriglandular disturbances (II and III) and other diseases (IV). In each group, three patients were found who were entirely negative in all aspects of the physical examinations (V).

TABLE IV

50 PATIENTS WITH ROOT RESORPTIONS AND WITH PREVIOUS ORTHODONTIC TREATMENT

NO.	SYSTEMIC DISTURBANCE	AGE	SEX	B.M.R.	NO.	SYSTEMIC DISTURBANCE	AGE	SEX	B.M.R.
1	Hypothyroidism	15	♀	−20.0	8	Hypothyroidism plus hypogonadism	23	♂	−18.0
2	Hypothyroidism	15	♀	−26.3					
4	Hypothyroidism	15½	♀	−15.4	10	Hypothyroidism plus hypogonadism	21	♂	−24.8
5	Hypothyroidism	24	♀	−10.0					
7	Hypothyroidism	17	♀	−20.0	15	Hypothyroidism plus hypogonadism	25	♀	−25.0
12	Hypothyroidism	15	♂	−17.0					
16	Hypothyroidism	23	♀	−23.7	37	Hypothyroidism plus hypogonadism	25	♀	−16.4
18	Hypothyroidism	14	♀	−21.0					
20	Hypothyroidism	23	♂	−26.0	40	Hypothyroidism plus hypogonadism	16	♂	−13.7
27	Hypothyroidism	19	♀	+ 5.0					
31	Hypothyroidism	27	♀	+ 9.0	41	Hypothyroidism plus hypogonadism	21	♂	−16.8
34	Hypothyroidism	34	♂	−11.3					
36	Hypothyroidism	21	♀	+ 0.5	71	Hypothyroidism plus hypogonadism	26	♂	− 2.3
38	Hypothyroidism	15	♀	+ 9.0					
39	Hypothyroidism	19	♀	−26.8	57	Hypothyroidism plus hyperparathyroidism	22	♂	−25.0
43	Hypothyroidism	11	♀	− 2.6					
45	Hypothyroidism	18	♀	−17.6	32	Hyperthyroidism	33	♂	+17.0
48	Hypothyroidism	20	♀	−27.4	68	Hyperthyroidism plus hypogonadism and hypopituitarism	21	♀	+46.0
59	Hypothyroidism	23	♂	−15.2					
60	Hypothyroidism	14	♀	−15.8					
61	Hypothyroidism	30	♂	+12.0	42	Hyperpituitarism	35	♂	+13.0
64	Hypothyroidism	21	♂	−13.2	50	Hypopituitarism	23	♀	− 2.2
69	Hypothyroidism	19	♀	−17.0	44	Hypopituitarism plus hypogonadism	15	♀	+ 5.5
70	Hypothyroidism	36	♀	− 9.3					
72	Hypothyroidism	21	♂	−13.8	82	Hypogonadism	22	♀	+ 1.2
77	Hypothyroidism	14	♀	−20.2	6	Series of acute and chronic infectious diseases	18	♂	− 5.4
79	Hypothyroidism	15	♀	−10.8					
80	Hypothyroidism	20	♂	−20.0					
81	Hypothyroidism	23	♀	−15.5	76	Negative physical examination	33	♂	−10.2
83	Hypothyroidism	23	♀	−					
9	Hypothyroidism plus hypopituitarism	21	♀	−10.2	75	Negative physical examination	20	♂	− 9.8
3	Hypothyroidism plus hypogonadism	18	♀	−10.0	78	Negative physical examination	19	♂	± 0.0

Table III gives the percentage of endocrine involvement and systemic diseases *other* than endocrine unbalance and shows the surprising result that the total of all systemic diseases on each side amounts to exactly 94 per cent (total of I to IV). There is, however, a different percentage of the various types. Even though we are not justified as yet to state that these various diseases have *produced* root resorptions—but rather only that they were found in *coexistence* with them—this high frequency seems to indicate that they determine the individual susceptibility to it.

The basal metabolic rate, determined routinely in most patients, is recorded in Tables IV and V; these indicate the high frequency of low basal rates in both groups.

TABLE V

50 PATIENTS WITH ROOT RESORPTIONS AND WITHOUT PREVIOUS ORTHODONTIC TREATMENT

NO.	SYSTEMIC DISTURBANCE	AGE	SEX	B.M.R.	NO.	SYSTEMIC DISTURBANCE	AGE	SEX	B.M.R.
14	Hypothyroidism	45	♀	–32.0	95	Hypothyroidism plus hypogonadism	38	♀	–14.2
17	Hypothyroidism	22	♂	–25.0					
20	Hypothyroidism	23	♂	–26.0	100	Hypothyroidism plus hypogonadism	41	♀	– 7.2
22	Hypothyroidism	60	♀	– 2.6					
23	Hypothyroidism	34	♀	+ 8.4	89	Hypothyroidism plus hypogonadism and hypopituitarism	28	♀	–20.5
30	Hypothyroidism	30	♀	–13.6					
46	Hypothyroidism	43	♀	–15.9					
56	Hypothyroidism	14	♂	+11.9	96	Hypothyroidism plus hypogonadism and hypopituitarism	30	♀	+ 4.4
61	Hypothyroidism	30	♂	+12.0					
83	Hypothyroidism	23	♀	–					
90	Hypothyroidism	20	♀	–	108	Hypothyroidism plus hypogonadism and hypopituitarism	44	♀	–14.3
91	Hypothyroidism	25	♀	–10.0					
99	Hypothyroidism	26	♀	–18.0					
101	Hypothyroidism	16	♂	– 6.2	29	Hyperthyroidism	12	♀	+34.0
102	Hypothyroidism	14	♀	+ 2.0	94	Hyperthyroidism	36	♂	+ 2.7
103	Hypothyroidism	32	♂	–	11	Hyperpituitarism	35	♀	– 1.7
109	Hypothyroidism	11	♀	–15.1	62	Hyperpituitarism	33	♂	– 5.7
111	Hypothyroidism	27	♀	–12.5	86	Hyperpituitarism	25	♀	– 6.0
112	Hypothyroidism	24	♀	–11.2	105	Hyperpituitarism	33	♂	–
114	Hypothyroidism	28	♂	–	107	Hypogonadism	26	♂	– 6.0
52	Hypothyroidism plus hypopituitarism	41	♀	– 9.0	28	Hypertension and kidney disease	46	♂	+19.1
13	Hypothyroidism plus hypogonadism	22	♀	–17.0	35	Duodenal ulcer	45	♂	+ 0.7
					87	Severe intestinal disturbance	30	♂	–
19	Hypothyroidism plus hypogonadism	35	♂	–16.0					
					92	Purpura hemorrhagica	22	♀	–
21	Hypothyroidism plus hypogonadism	53	♂	–12.7	98	Secondary anemia, amebic dysentery	37	♂	– 4.0
41	Hypothyroidism plus hypogonadism	21	♂	–16.8	104	Tuberculosis? Dietary deficiency	19	♂	– 3.1
58	Hypothyroidism plus hypogonadism	20	♀	–10.0	113	Arthritis	24	♂	– 7.0
					93	Neg. physical exam.	20	♂	– 1.9
63	Hypothyroidism plus hypogonadism	18	♀	–10.9	97	Neg. physical exam. (Negro)	38	♀	– 5.0
88	Hypothyroidism plus hypogonadism	33½	♀	+12.4	110	Neg. physical exam.	13	♀	+ 4.0

TABLE VI

BASAL METABOLIC RATE AND CHOLESTEROL CONTENT OF BLOOD PLASMA IN 16 CASES OF ROOT RESORPTION FOUND IN COEXISTENCE WITH HYPOTHYROIDISM AND HYPOTHYROIDISM PLUS OTHER ENDOCRINOPATHIES

NO.	SEX	AGE	ENDOCRINE DISTURBANCE	B.M.R.	CHOLESTEROL MG. %
8	♂	23	Hypothyroidism plus hypogonadism	– 18.0	214
10	♂	21	Hypothyroidism plus hypogonadism	– 18.8	238
18	♀	14	Hypothyroidism	– 21.0	188
20	♂	23	Hypothyroidism	– 26.0	159
39	♀	19	Hypothyroidism	– 26.8	208
57	♂	22	Hypothyroidism plus hyperparathyroidism	– 25.0	190
60	♀	14	Hypothyroidism	– 15.8	176
69	♀	19	Hypothyroidism	– 17.0	225
72	♂	21	Hypothyroidism	– 13.8	220
79	♀	15	Hypothyroidism	– 10.8	208
81	♀	23	Hypothyroidism	– 15.5	214
91	♀	25	Hypothyroidism	– 10.0	231
95	♀	38	Hypothyroidism plus hypogonadism	– 14.2	205
99	♀	26	Hypothyroidism	– 18.0	190
111	♀	27	Hypothyroidism	– 12.5	163
112	♀	24	Hypothyroidism	– 11.2	226

Since the diagnosis of hypothyroidism meets so frequently with considerable difficulties, determinations of blood cholesterol were made and compared with the basal metabolic rate and clinical symptoms (Table VI).

It may be noted that with three exceptions (Nos. 20, 60, 111) the low basal metabolic rate was accompanied by a high cholesterol content of blood plasma. (Normal values range between 150 and 180 mg. per cent, according to Mandel-Steudel.) If all other diseases which are accompanied frequently by cholesteremia, as for instance diabetes, xanthomatosis, acute leucemia, acne vulgaris, psoriasis, etc., can be excluded from a differential diagnostic standpoint, it is believed that this test can be used to great advantage as an aid in the diagnosis of hypothyroidism. (For further discussion, see under Comment.)

Independent of the physical examinations made of these patients the roentgenograms of the teeth and jaws were interpreted and classified as to their atrophic, dystrophic, or normal bone conditions (Tables VII and VIII).

This survey also presents an extremely interesting and important result. The group with previous orthodontic treatment shows a frequency of 74 per

TABLE VII

FREQUENCY OF ATROPHIC AND DYSTROPHIC BONE CHANGES FOUND IN COEXISTENCE WITH SYSTEMIC DISTURBANCES IN 100 PATIENTS WITH ROOT RESORPTIONS

		50 CASES WITH PREVIOUS ORTHODONTIC TREATMENT						50 CASES WITHOUT PREVIOUS ORTHODONTIC TREATMENT					
				BONE						BONE			
				ATROPHIC OR DYSTROPHIC		NORMAL				ATROPHIC OR DYSTROPHIC		NORMAL	
		NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT
I	Hypothyroidism	30	60.0	21	70.0	9	30.0	20	40.0	19	95.0	1	5.0
II	Hypothyroidism and other endocrine disturbances	10	20.0	8	80.0	2	20.0	13	26.0	12	92.3	1	7.7
	Total: I and II	40	80.0	29	72.5	11	27.5	33	66.0	31	93.9	2	6.1
III	Endocrine disturbances without hypothyroidism	6	12.0	5	83.3	1	16.7	7	14.0	6	85.7	1	20.0
	Total: I, II, and III	46	92.0	34	73.9	12	26.1	40	80.0	37	92.5	3	7.5
IV	Systemic disturbances other than endocrine unbalance	1	2.0	1	100.0	–	–	7	17.0	7	100.0	–	–
	Total: I, II, III, and IV	47	94.0	35	74.4	12	25.6	47	94.0	44	93.6	3	6.4
V	Negative physical examination	3	6.0	2	66.6	1	33.4	3	6.0	3	100.0	–	–
	Total	50	100.0	37	74.0	13	26.0	50	100.0	47	94.0	3	6.0

cent atrophic and dystrophic bone lesions, while the other group which had not been treated orthodontically shows 20 per cent higher involvement of the osseous structure, i.e., exactly 94 per cent. These comparative figures indicate that the extremely high frequency of atrophic and dystrophic bone lesions in the fifty patients who had *not* been treated orthodontically seems to be related to the resorptive processes of the root ends. In the other group we must account for the 20 per cent higher frequency of normal bone, which according to our contention should not have led to root resorptions. Since we have every reason to believe that the individual susceptibility to root resorptions depends, to a large extent, on the individual disposition to resorptive activity, which in turn depends on certain endogenous factors, there seems to be no other explanation at the present time but that a mechanical factor in these 20 per cent, i.e., a tremendous orthodontic force, has led to the disappearance of roots (pressure atrophy). In most instances this was confirmed by the patient or his orthodontist. From a practical standpoint, it means that in all cases under our observation only 20 per cent, or one-fifth, which develop root resorptions do so under orthodontic treatment on the basis of too much pressure. The others probably would develop it anyway, as the result of the factors producing pathologic bone formation, regardless of whether treated orthodontically or not.

TABLE VIII

ROENTGENOGRAPHIC EVIDENCE OF PATHOLOGIC BONE FORMATION IN 100 CASES OF ROOT RESORPTION

	50 CASES WITH PREVIOUS ORTHODONTIC TREATMENT					50 CASES WITHOUT PREVIOUS ORTHODONTIC TREATMENT				
	NUMBER OF CASES	BONE				NUMBER OF CASES	BONE			
		ATROPHIC OR DYSTROPHIC		NORMAL			ATROPHIC OR DYSTROPHIC		NORMAL	
		CASES	PER CENT	CASES	PER CENT		CASES	PER CENT	CASES	PER CENT
Series I*	35	25	71.4	10	28.6	19	17	89.4	2	10.6
Series II	15	12	80.0	3	20.0	31	30	96.7	1	3.3
Total	50	37	74.0	13	26.0	50	47	94.0	3	6.0

*Series I has been reported in the J. Dent. Res. 14: June, 1934.

In a further attempt to uncover in these patients whether a dietary deficiency of a longer period of time occurs simultaneously with root resorption, the possibility of which was suggested by the animal experimentation mentioned in the beginning, the nutritional habits of each group were studied and carefully analyzed by Dr. Simmonds (Tables IX and X).

In these studies attention was given to the amount of intake of all food constituents with special reference to calcium, phosphorus, vitamins C and D necessary for the development of teeth and bone, during three periods of life, i.e., between birth and ten years, between ten and twenty years, and over twenty years of age to the present time. The values entered on Tables IX and X are given for each patient as excellent, excellent to good, fair to good, fair, low to fair, and low.

Tables IX and X indicate that by and large the nutrition of the 20 patients who had previous orthodontic treatment is considerably better than that found

in the other group. Among the former we find 5 patients (Cases 18, 77, 80, 10, and 82) who had up to the present time no low or low to fair values for Ca, P, vitamin C or D, while in the latter we find only one case (Case 89). The total low to fair and low values in the group without previous orthodontic treatment amount to 68, against only 54 in the other group.

TABLE IX

NUTRITIONAL ANALYSIS OF 20 PATIENTS WITH ROOT RESORPTIONS AND WITH PREVIOUS ORTHODONTIC TREATMENT*

CASE NO.		AGE	BIRTH-10 YEARS				10-20 YEARS				20 YEARS TO PRESENT			
			Ca	P	VIT. C	VIT. D	Ca	P	VIT. C	VIT. D	Ca	P	VIT. C	VIT. D
4	Hypothyroidism	16	E	E	F	L	GE	GE	GE	?	–	–	–	–
18	Hypothyroidism	15	G	G	G	G	GE	GE	G	G	–	–	–	–
64	Hypothyroidism	22	GE	GE	L	L	E	E	GE	G	–	–	–	–
69	Hypothyroidism	20	E	E	FG	L	GE	G	E	F	–	–	–	–
70	Hypothyroidism	37	E	E	G	LF	FG	E	G	LF	LF	EG	G	LF
72	Hypothyroidism	22	–	–	–	–	F	E	LF	L	–	–	–	–
77	Hypothyroidism	15	G	G	G	G	G	G	G	G	–	–	–	–
79	Hypothyroidism	16	FG	FG	LF	L	FG	G	FG	L	–	–	–	–
80	Hypothyroidism	18	FG	FG	G	G	G	F	FG	G	–	–	–	–
81	Hypothyroidism	27	FG	FG	FG	LF	F	LF	F	LF	L	FG	LF	L
8	Hypothyroidism plus hypogonadism	25	GE	E	L	G	F	E	L	LF	GE	E	G	E
10	Hypothyroidism plus hypogonadism	23	E	E	G	G	E	E	EG	F	E	E	G	F
45	Hypothyroidism plus hypogonadism	20	GE	GE	L	L	GE	GE	G	GF	–	–	–	–
71	Hypothyroidism plus hypogonadism	27	FG	G	LF	L	G	LF	LF	L	G	LF		
11	Hyperpituitarism	35	F	F	L	L	G	G	L	L	L	L	L	L
42	Hyperpituitarism	37	E	E	L	LF	GE	GE	L	LF	G	G	L	LF
82	Hypogonadism	28	G	G	G	F?	G	FG	G	F?	–	–	–	–
76	Negative physical examination	34	E	E	L	LF	GE	GE	L	LF	G	G	L	LF
78	Negative physical examination	21	G	G	L	L	G	E	G	LF	–	–	–	–
75	Negative physical examination	21	FG	E	LF	FG	E	G	E	G	–	–	–	–
	Excellent	(E)	6	8	–	–	3	6	2	–	1	2	–	1
	Excellent to good	(EG)	3	2	–	–	6	5	3	–	1	1	–	–
	Good	(G)	4	5	6	5	5	6	6	5	2	3	3	–
	Fair to good	(FG)	5	3	2	1	2	1	2	1	–	1	–	–
	Fair	(F)	1	1	1	1	3	1	1	4	–	–	–	1
	Low to fair	(LF)	–	–	3	4	1	1	2	7	1	–	2	4
	Low	(L)	–	–	7	8	–	–	4	2	3	1	3	2
	Low plus, low to fair		–	–	10	12	1	1	6	9	4	1	5	6
			Total of low and fair values = 54											

*A detailed discussion of the nutritional findings in these patients will be published separately by Dr. Nina Simmonds.

The vitamin C and D deficiency in both groups up to ten years of age is outstanding; while the number with a deficient calcium intake increases with advancing age. Whether endocrine disturbances have any influence on the individual selection of certain foodstuffs, as for instance the craving for sweets in diabetes, or whether the food intake influences the development of an endocrine imbalance is an extremely interesting problem. Large amounts of material and much experimental work are required to solve this, a project which is wholly beyond our field.

TABLE X

NUTRITIONAL ANALYSIS OF 20 PATIENTS WITH ROOT RESORPTIONS AND WITHOUT PREVIOUS ORTHODONTIC TREATMENT

CASE NO.		AGE	BIRTH-10 YEARS				10-20 YEARS				20 YEARS TO PRESENT			
			Ca	P	VIT. C	VIT. D	Ca	P	VIT. C	VIT. D	Ca	P	VIT. C	VIT. D
17	Hypothyroidism	22	E	E	E	F	G	G	E	G	F	G	F	L
23	Hypothyroidism	34	FL	F	F	?	L	F	F	L	–	–	–	–
56	Hypothyroidism	14	FG	GE	G	F	G	G	G	LF	–	–	–	–
90	Hypothyroidism	20	FG	FG	LF	L	L	LF	F	L	–	–	–	–
99	Hypothyroidism	26	G	G	L	L	G	G	L	F	G	G	LF	F
101	Hypothyroidism	16	G	GE	LF	LF	F	G	LF	LF	–	–	–	–
111	Hypothyroidism	27	E	GE	LF	FG	E	E	F	FG	G	G	FG	F
114	Hypothyroidism	24	E	E	LF	LF	FG	E	LF	F	FG	FG	FG	F
91	Hypothyroidism	25	–	–	–	–	–	–	–	–	LF	FG	L	F
19	Hypothyroidism plus hypogonadism	35	G	G	LF	F	F	F	LF	L	G	G	F	L
100	Hypothyroidism plus hypogonadism	41	G	G	L	L	G	G	L	F	L	G	F	L
89	Hypothyroidism plus hypogonadism and hypopituitarism	28	E	E	G	G	E	E	G	G	E	E	G	G
96	Hypothyroidism plus hypogonadism and hypopituitarism	30	G	G	FG	LF	F	F	G	G	L	F	G	L
11	Hyperpituitarism	35	F	F	L	L	G	G	L	L	L	L	L	L
35	Other diseases	45	LF	G	L	L	L	E	L	L	L	E	LF	L
87	Other diseases	30	LF	FG	L	L	F	G	L	LF	LF	FG	LF	L
97	Other diseases	38	E	E	LF	G	GE	E	G	FG	G	E	GE	F
93	Negative physical examinations	20	G	G	F	L	G	G	FG	F	–	–	–	–
104	Negative physical examination	19	FG	FG	L	L	F	G	LF	LF	–	–	–	–
110	Negative physical examination	13	E	E	LF	F	–	–	–	–	–	–	–	–
	Excellent	(E)	6	5	1	–	2	5	1	–	1	3	–	–
	Excellent to good	(EG)	–	3	–	–	1	–	–	–	–	–	1	–
	Good	(G)	6	6	2	2	6	9	4	3	4	5	2	1
	Fair to good	(FG)	3	3	1	1	1	–	1	2	1	3	2	–
	Fair	(F)	1	2	2	4	5	3	3	4	1	1	3	5
	Low to fair	(LF)	3	–	7	3	–	1	4	4	2	–	3	–
	Low	(L)	–	–	6	8	3	–	5	5	4	1	2	7
	Low plus, low to fair		3	–	13	11	3	1	9	9	6	1	5	7

Total of low and fair values = 68

On the basis of roentgenographic evidence 773 definite root resorptions were observed in the group *with* and 733 in the group *without* orthodontic treatment. The difference is not very marked, being only about 5 per cent. Since it is difficult in many instances to determine roentgenographically whether the peculiar appearance of a root end is the result of a resorptive activity or of a developmental deficiency, a large number of teeth were recorded in Tables XI, XII, and XIII as being questionable.

There are 184 "questionable resorptions" in Table XI and 111 in Table XII, which give a total of 957 resorptions in the group with previous orthodontic treatment and 844 without, a difference of less than 12 per cent.

This survey shows very definitely that there is little difference in the number of resorptions as affecting the various teeth in both groups, especially if we consider that three patients of the group without previous orthodontic

TABLE XI

ROOT RESORPTIONS OBSERVED IN 50 PATIENTS WITH PREVIOUS ORTHODONTIC TREATMENT

TEETH	MAXILLA		MANDIBLE		MAXILLA AND MANDIBLE		MAXILLA AND MANDIBLE	
	RESORPTIONS		RESORPTIONS		RESORPTIONS		SINGLE-ROOTED TEETH	MULTI-ROOTED TEETH
	DEFINITE	?	DEFINITE	?	DEFINITE	?		
Central incisors	94	1	98	–	192	1		
Lateral incisors	87	4	90	3	177	7		
Cuspids	65	23	67	8	132	31	687 (132)*	
First bicuspids	54	23	50	20	104	43		
Second bicuspids	40	27	42	23	82	50		
Molars	29	28	57	24	86	52		86 (52)*
Total number of resorptions	369	106	404	78	773	184		
Total	475		482		957			

*Questionable resorptions.

TABLE XII

ROOT RESORPTIONS OBSERVED IN 50 PATIENTS WITHOUT PREVIOUS ORTHODONTIC TREATMENT

	MAXILLA*		MANDIBLE		MAXILLA AND MANDIBLE		MAXILLA AND MANDIBLE	
	RESORPTIONS		RESORPTIONS		RESORPTIONS		SINGLE-ROOTED TEETH	MULTI-ROOTED TEETH
	DEFINITE	?	DEFINITE	?	DEFINITE	?	RESORPTIONS	
Central incisors	82	4	84	4	166	8		
Lateral incisors	77	5	79	6	156	11		
Cuspids	55	13	59	12	114	25	662 (86)†	
First bicuspids	54	16	58	10	112	26		
Second bicuspids	55	11	59	5	114	16		
Molars	26	13	45	12	71	25		71 (25)†
Total number of resorptions	349	62	384	49	733	111		
Total	411		433		844			

*Among these patients three had edentulous maxillas.
†Questionable resorptions.

TABLE XIII

INCIDENCE OF ROOT RESORPTION OF THE DIFFERENT TEETH OF 100 PATIENTS

TEETH	ROOT RESORPTIONS IN 50 PATIENTS WITH PREVIOUS ORTHODONTIC TREATMENT		ROOT RESORPTIONS IN 50 PATIENTS WITHOUT PREVIOUS ORTHODONTIC TREATMENT	
	DEFINITE	?	DEFINITE	?
Central incisors	192	1	166	8
Lateral incisors	177	7	156	11
Cuspids	132	31	114	25
First bicuspids	104	43	112	26
Second bicuspids	82	50	114	16
Molars	86	52	71	25
Total	773	184	733	111

treatment had edentulous maxillas and no roentgenograms could be obtained, which were made before the extraction of the maxillary teeth. One thing, however, seems to stand out in both groups, and that is that the highest frequency of resorptions occurs in the central incisors and that the other teeth follow in the sequence as listed in Tables XI, XII, and XIII. And furthermore, it can be noted that the frequency of resorptions of the mandibular molars is definitely higher than that of the same teeth in the maxilla. These findings are in direct contrast with the statement of Schmid, that molars and premolars *never* show any resorptions. There is also a higher frequency of resorption on single-rooted than on multirooted teeth (Tables XI and XII).

The various statements as to a possible influence of types of appliances on resorptions of the root ends were also checked in this material. A correlation as established by Ketcham, however, could not be found. In some instances it was not possible to obtain exact information as to methods of treatment. For this reason the reports of only 42 patients were evaluated (Table XIV).

Table XIV indicates that patients treated with the lingual arch have less resorption than those treated with other appliances. The number of observations, however, is far too small to allow for any definite conclusion. It is interesting to note that the calculation of the average number of resorptions per patient reveals (last column) 12 resorptions for the lingual arch group, 13 for the pin and tube, 14 for the labial arch, and 15 for the ribbon arch.

As to the influence of severe loosening and "jiggling" of teeth during the course of orthodontic treatment, it was found that of the 50 patients treated orthodontically, severe loosening and jiggling were denied in 38 instances, and only 19 patients complained about pain as the result of treatment.

TABLE XIV

TYPES OF APPLIANCES USED IN 42 PATIENTS WITH ROOT RESORPTIONS

TYPE OF APPLIANCE	MAXILLARY TEETH			MANDIBULAR TEETH			TOTAL NUMBER OF JAWS TREATED	TOTAL NUMBER OF RESORPTIONS IN MAXILLAS AND MANDIBLES		AVERAGE NUMBER OF RESORPTIONS PER PATIENT
	NUMBER OF JAWS TREATED	NUMBER OF TEETH SHOWING RESORPTIONS		NUMBER OF JAWS TREATED	NUMBER OF TEETH SHOWING RESORPTIONS					
		DEFINITE	?		DEFINITE	?		DEFINITE	?	
Lingual	8	52	16	10	54	27	18	106	43	12
Pin and tube	11	76	26	9	55	4	20	131	30	13
Labial	12	86	39	12	86	17	24	172	56	14
Ribbon bracket	9	56	24	13	104	34	22	160		15
Total	40	270	104	44	299	82	84	596	187	

The statement of Oppenheim that resorptions are found mainly following individual tooth movement in Class I cases, and that resorptions are rarely found where individual tooth movement was not performed—as, for instance, in Class II and III cases—could not be supported by our findings (Table XV). Table XV shows that there is very little difference in the number of resorptions following the treatment of a Class I or Class II anomaly. Because of the smaller number of patients diagnosed as Class III, there are fewer resorptions in this

TABLE XV

NUMBER OF ROOT RESORPTIONS IN 48 PATIENTS CLASSIFIED AS TO THE ORTHODONTIC DIAGNOSIS BEFORE BEGINNING OF TREATMENT

	NUMBER OF PATIENTS		RESORPTIONS							AVERAGE NUMBER PER PATIENT
			INCISORS		CUSPIDS	BI-CUSPIDS	MOLARS	TOTAL		
	TREATED	TOTAL	CENTRAL	LATERAL						
Class I	11	21	42	38	34	31	15	160	333	16
Class I with symptoms of Class II	10		40	36	28	43	26	173		
Class II, Subdivision 1	16	22	69	56	39	57	20	241	318	14
Class II, Subdivision 2	6		23	23	10	16	6	77		
Class III	5	5	18	17	14	22	9	80		16
Total	48		192	170	125	169	75			

TABLE XVI

AGE AT BEGINNING OF ORTHODONTIC TREATMENT, NUMBER OF RESORPTIONS FOUND, AND DURATION OF TREATMENT

BEGINNING OF ORTHODONTIC TREATMENT		NUMBER OF TEETH SHOWING RESORPTIONS		AVERAGE NUMBER OF RESORPTIONS PER PATIENT	NUMBER OF YEARS' DURATION OF ORTHODONTIC TREATMENT														
AGE	NUMBER OF PATIENTS	DEFINITE	?		1 MO.	½	1	1½	2	2½	3	3½	4	4½	5	7	8	10	12
7	4	67	11	17										1				2	1
8	2	24	7	12												1	1		
9	3	21	11	7			1				1				1				
10	6	92	17	15		1			1						3	1			
11	1	9	5	9	1														
12	9	146	36	16		1	1	1		1	2	1	1		1				
13	8	133	19	17			3		2		2				1				
14	5	79	31	16							1		2	1	1				
15	2	33	5	16									1			1			
16	3	43	20	14							1		1			1			
18	1	17	–	17									1						
19	1	14	7	14				1											
20	3	62	5	21				1			1		1						
21	1	22	–	22					1										
22	1	11	10	11			1												
Total	50	773	184		1	2	6	3	4	1	8	1	7	2	7	4	1	2	1

group. A calculation of the average number of resorptions per patient, however, reveals 16 for Class I, 14 for Class II, and 16 for Class III.

Since it has been stated at different times that the age of the patient at the beginning of orthodontic treatment is of definite importance in the production of root resorption, this was checked and recorded in Table XVI.

It can be seen from Table XVI that neither the age at the beginning of orthodontic treatment nor the number of years' duration can be held responsible for the incidence of root resorptions. This can be seen in the column, "Average Resorptions Per Patient."

COMMENT

On the basis of the material presented it can be readily seen that the problem of root resorption is one which involves the medical as well as the dental profession. The high frequency of systemic diseases (94 per cent) in patients requiring orthodontic treatment, in whom root resorptions were later found, indicates that now more than ever we must familiarize ourselves with the biologic factors which may influence the result of orthodontic treatment. I am decidedly of the opinion that the root resorptions observed in orthodontic practice are produced not by mechanical force *alone*, but that they are rather the result of an individual predisposition to increased resorptive activity, we might say, when a very "labile" osseous structure is found which in turn is due to various endogenous factors.

I. THYROID DYSFUNCTION

Among the endocrine disturbances hypothyroidism seems to occupy a dominant place. With regard to its influence on the osseous structure it is well to keep in mind that the thyroid gland functions normally to maintain cellular nutrition and growth and that under hypothyroid conditions these processes are inhibited (Janney). The very high frequency of atrophic and dystrophic bone lesions in patients with hypothyroidism suggests that the normal process of bone formation—the balance between resorption and deposition—is severely disturbed. A parallel investigation which is being carried on at the present time to determine the frequency of systemic involvement in a certain type of paradentosis, which I have designated as the "osteopathic type," has brought to light that a high percentage of patients who were suffering from this type of paradentosis also had simultaneously a thyroid disturbance with a primary involvement of the osseous structure.

Kunde and Carlson (1927) demonstrated that thyroidectomized rabbits, showing signs of hypothyroidism during the growth period, manifested a defective calcification and growth of the bones simulating clinical rickets. It seems, therefore, to indicate that the formation of pathologic bone in these animals is part of an osteodystrophic condition, characterized by the formation of a deficient osseous structure.

Through the work of Engelbach and McMahon, Shelton and others, it is well established that the retardation of osseous development is pathognomonic of early hypothyroidism. Shelton in particular points out that "comparatively few hypothyroid children present the classic evidence of cretinism or myxedema unless the condition is far advanced. As a consequence, many are considered

normal until some outspoken evidence of the thyroid deficiency is presented in later life.'' This statement contains much truth. In our experience, even with competent medical men, we quite frequently find that they have failed to recognize the early diagnosis of such a condition or at least have paid very little attention to its obvious symptoms.

To avoid any misunderstanding, it should be stated that in the course of this investigation the diagnosis of hypothyroidism was made *on the basis of definite clinical symptoms and laboratory tests, which included the basal metabolic rate, cholesterol determination, and differential blood count.* In patients under fifteen years of age the bone age was determined as a valuable aid in the diagnosis.

The high frequency of hypothyroidism thus established in patients with root resorptions, which amounts to 60 per cent in the treated group and 40 per

Fig. 15.

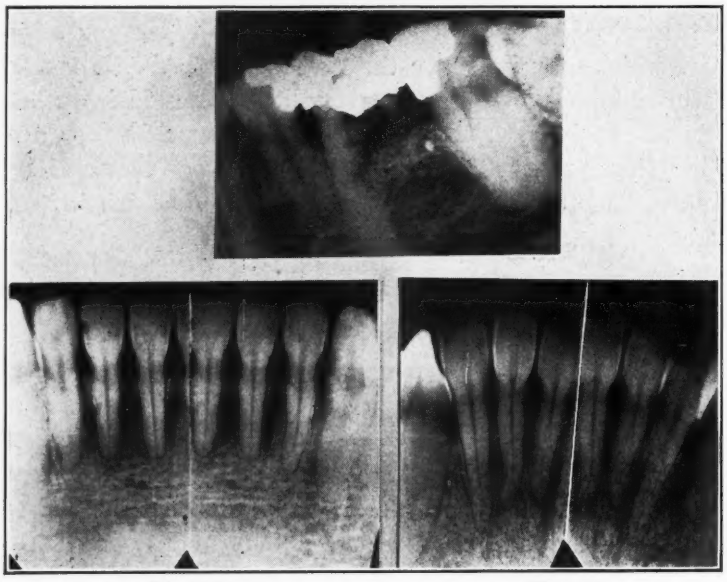

Fig. 16. Fig. 17.

Fig. 15.—Complete resorption of distal root of left mandibular first molar with large resorptive area of the mesial root. No previous orthodontic treatment. Aged forty-three years; systemic disease: hypothyroidism. (Patient No. 46.)

Fig. 16.—Root resorptions of anterior teeth accompanied by interdental osteoporotic lesion. No previous orthodontic treatment. Aged thirty years; systemic disease: hypothyroidism. (Patient No. 30.)

Fig. 17.—Root resorptions with apical widening of the peridental membrane space. No previous orthodontic treatment. Aged forty-five years; systemic disease: hypothyroidism. (Patient No. 14.)

cent in the group without orthodontic treatment, not only is surprising but seems to carry a particular significance. Since hypothyroidism is frequently associated with, or perhaps even the result of, other endocrinopathies, it seemed well to add this group (II, Table III) to the former, which brings the frequency of hypothyroidism alone and hypothyroidism plus other endocrine disturbances up to 80 per cent in the group *with* and 66 per cent in the group *without* previous orthodontic treatment. All endocrinopathies other than hypothyroidism as listed under III in Table III seem to play only a minor rôle.

In charting the atrophic and dystrophic bone changes against hypothyroidism and hypothyroidism with other endocrine disturbances, we find that these

two groups are associated with pathologic bone formation very frequently, in fact in 72.5 per cent of the group with, and in 93.9 per cent of the group without orthodontic treatment (Table VII). The 20 per cent difference between the two groups may be explained by the fact that the age of the patients who have had no orthodontic treatment is considerably higher, on an average, than that in the first group. In other words, it may be possible that increasing age, with a number of accompanying factors, not only has a definite influence on the osseous structure (senile osteoporosis) but also involves the tooth structure whether orthodontic treatment has been performed or not.

Fig. 18. Fig. 19.

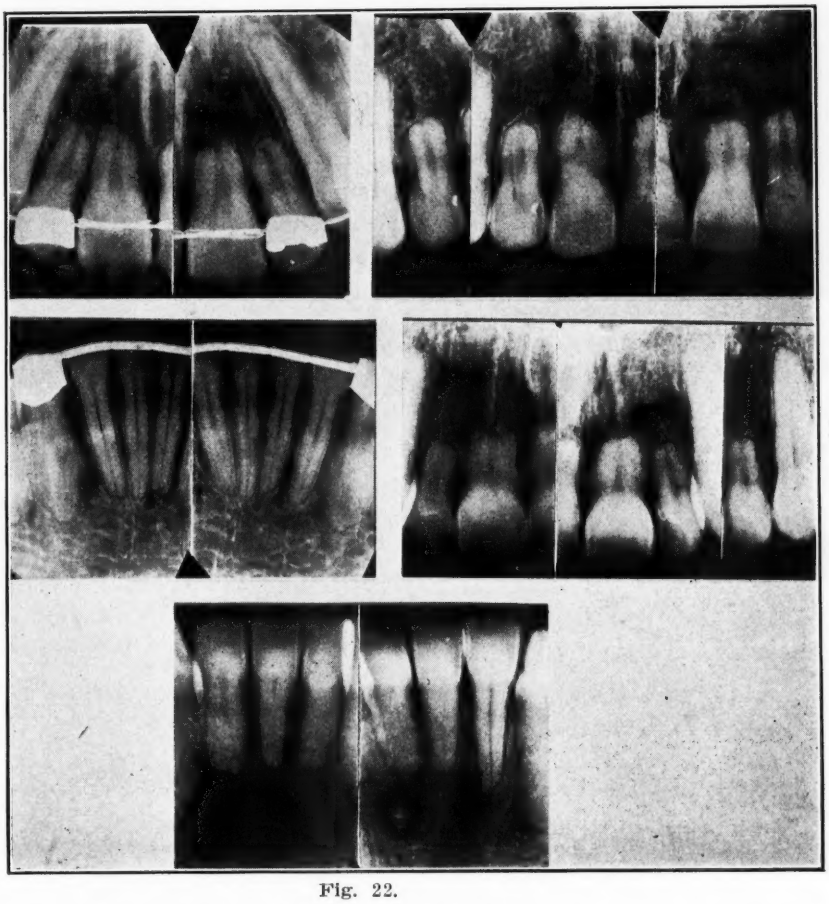

Fig. 20. Fig. 21.

Fig. 22.

Fig. 18.—Root rescrptions following orthodontic treatment for three years. Aged 15 years; systemic disease: hypothyroidism. (Patient No. 12.)

Fig. 19.—Advanced root resorptions of anterior and posterior teeth following four years of orthodontic treatment. Apical peridental membrane space has again reached normal width suggesting arrest of the resorptive process. Note extreme osteoporotic lesion over apices. Aged thirty years; systemic disease: hypothyroidism. (Patient No. 61.)

Fig. 20.—Resorption of roots of mandibular anterior teeth accompanied by osteoporotic lesion and slight horizontal atrophy. Aged thirty years. (Patient No. 61.)

Figs. 21 and 22.—Marked resorptions in maxilla and mandible following orthodontic treatment for seven years. Enlarged circulatory channels in mandibular anterior region with pathologic bone formation. Aged thirty-six years; systemic disease: hypothyroidism. (Patient No. 70.)

From the standpoint of bone pathology we must keep in mind that those changes, which we see roentgenographically in the jaws, accompanying hypothyroidism or any other systemic disease can also be the result of delayed

development instead of a pathologic process which has developed after completion of the formation of maxilla and mandible. This differentiation is not always very easy to make and requires a correlation of all clinical and laboratory findings, as well as an evaluation of nutritional habits and of the medical records with special reference to development in early childhood. It hardly needs to be mentioned that the diagnosis of these pathologic bone lesions requires excellent roentgenograms. It has been our experience that the majority of roentgenograms referred to us for diagnosis were unsuitable for this purpose.

If we assume that a thyroid deficiency has a very definite influence on bone and tooth development during early childhood, i.e., during the juvenile, infantile, and adolescent periods, which might lead clinically to enlarged mar-

Fig. 23. Fig. 24.

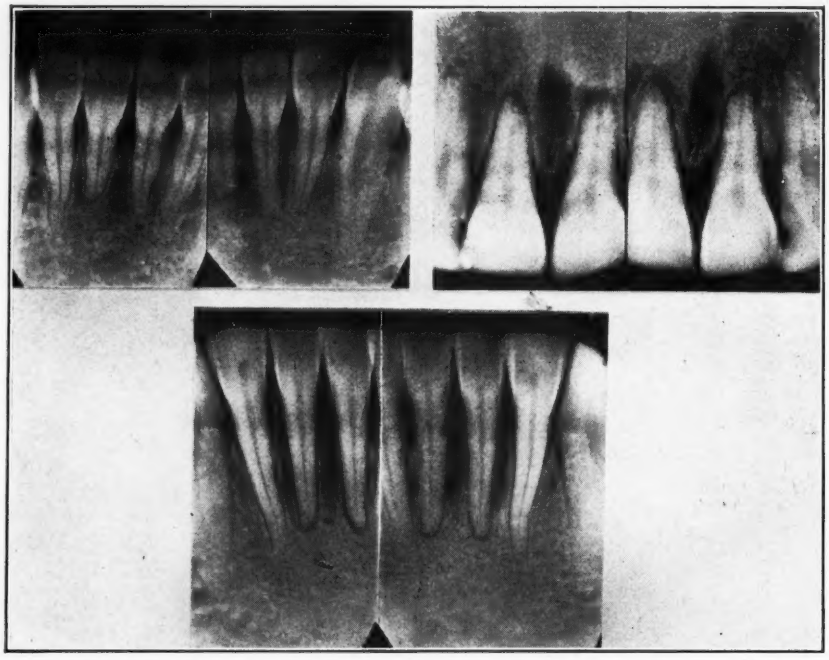

Fig. 25.

Fig. 23.—Multiple root resorptions accompanied by osteoporosis. No previous orthodontic treatment. Aged eighteen years; systemic disease: hypothyroidism and hypogonadism. (Patient No. 63.)

Figs. 24 and 25.—Resorptions in maxilla and mandible with peculiar fuzzy appearance of osseous structure. No previous orthodontic treatment. Aged forty-one years; systemic disease: hypothyroidism and hypogonadism. (Patient No. 100.)

row cavities in the jaws and the osseous part of the paradentium, it now becomes the duty of the orthodontic profession to learn how to interpret roentgenograms and especially to diagnose osseous changes. In the future this question must occupy a major place in the diagnostic procedure, especially of those practitioners living in goiter belts; although we must keep in mind that with a shifting population this condition can be found in any section of the country (Shelton). I remember that my first thought, after having calculated the high incidence of thyroid deficiencies occurring with root resorptions, was whether the reports published by Ketcham came from a goiter belt. It is well known that Denver and the Colorado River district are goiter centers, and the reports of Ketcham, in which he gives medical histories, seem to indicate quite clearly the presence of thyroid disturbances in some of his cases.

Figs. 15, 16, and 17* illustrate root resorptions found in coexistence with hypothyroidism in cases which had *not* been treated orthodontically.

Similar resorptions, however, are found in patients suffering from the same disturbance but *with* previous orthodontic treatment (Figs. 18-22).

II. HYPOTHYROIDISM PLUS OTHER ENDOCRINE DISTURBANCES

Figs. 23, 24, and 25 illustrate root resorptions found in coexistence with hypothyroidism and other endocrine disturbances in patients who have had *no* orthodontic treatment.

Fig. 26.

Fig. 27.

Fig. 28.

Figs. 26, 27, and 28.—Multiple resorptions in maxilla and mandible. Patient was physically underdeveloped with definite endocrine background. (Hypothyroidism and hypogonadism.) Marked bone atrophy in all parts of the jaws. Orthodontic treatment for ten years. Aged eighteen years. (Patient No. 3.)

Fig. 29.

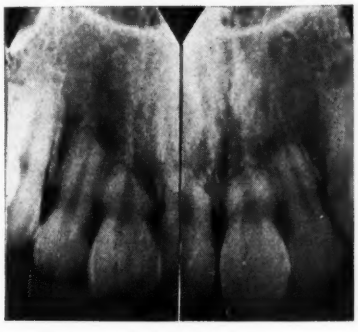

Fig. 30.

Fig. 29.—Root resorptions following four years' orthodontic treatment. Aged twenty-one years; systemic disease: hypothyroidism and hypogonadism. (Patient No. 10.)

Fig. 30.—Root resorption following seven years' orthodontic treatment. Note former lamina dura around apices. Aged twenty-one years; systemic disease: hypothyroidism and hypopituitarism. (Patient No. 9.)

Resorptions are also found in patients with the same disturbance but who *had* previous orthodontic treatment (Figs. 26-38).

III. ENDOCRINE DISTURBANCES OTHER THAN HYPOTHYROIDISM

Hyperpituitarism

At the beginning of our studies we had occasion clinically to examine an acromegalic person and to see the dental roentgenograms. We found that with-

*In the selection of roentgenograms for publication only cases showing marked lesions were chosen; these promised a good result in the reproduction. Because of limited space available, the number of pictures has been reduced to a minimum.

out exception all the roots were severely resorbed. This accidental finding led to an investigation of more cases of acromegaly, of which I can present five at the present time (Figs. 39-43).

Fig. 31. Fig. 32.

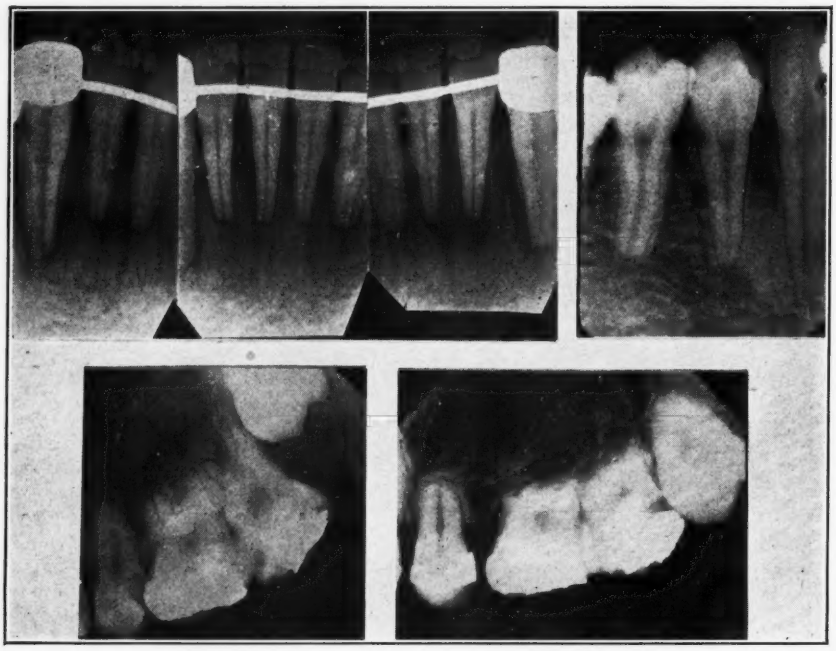

Fig. 33. Fig. 34.

Fig. 35. Fig. 36.

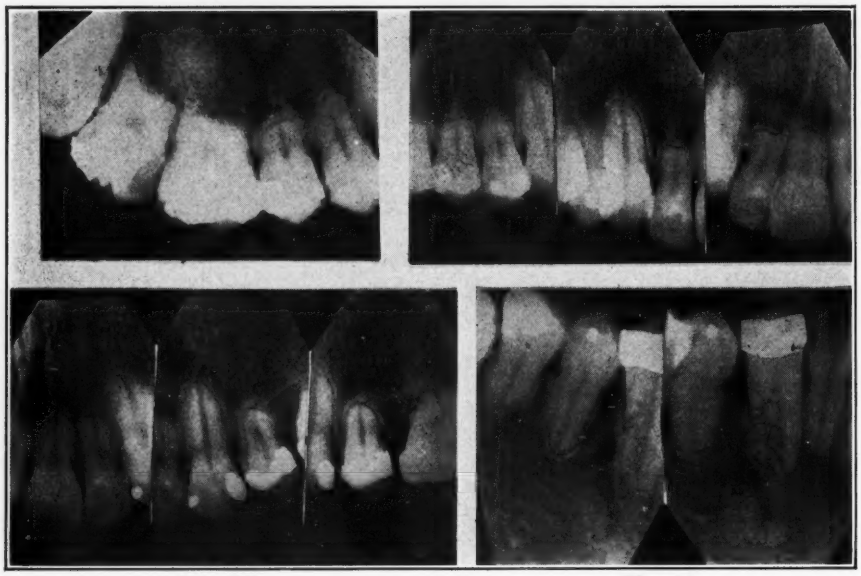

Fig. 37. Fig. 38.

Fig. 31.—Multiple resorptions during course of orthodontic treatment. Aged twenty-three years; systemic disease: hypothyroidism and hypogonadism. (Patient No. 8.)

Fig. 32.—Root resorptions and large pulp nodules following orthodontic treatment for one and one-half years. Aged twenty-one years; systemic disease: hypothyroidism and hypogonadism. (Patient No. 41.)

Figs 33-38.—Marked root resorptions following orthodontic treatment for five years. Bone has dystrophic appearance with irregularly calcified areas. Aged twenty-two years; systemic disease: hyperparathyroidism with thyroid dysfunction. (Patient No. 57.)

Figs. 39-43 demonstrate that all five patients with an active or inactive hyperpituitarism have generalized root resorption. Of these only one has been treated orthodontically (Fig. 43). This finding in particular confirms our

Fig. 39.—Multiple root resorptions found together with hyperpituitarism. (Acromegaly.) No previous orthodontic treatment. Aged thirty-three years. (Patient No. 11.)

original contention that the biologic aspect of the problem, i.e., the individual susceptibility to root resorption which is determined by endogenous factors surpasses by far the importance of the mechanical aspect.

According to P. Marie, the osseous changes in acromegaly consist in tremendous periosteal new formation of bone, with a disturbance in the balance

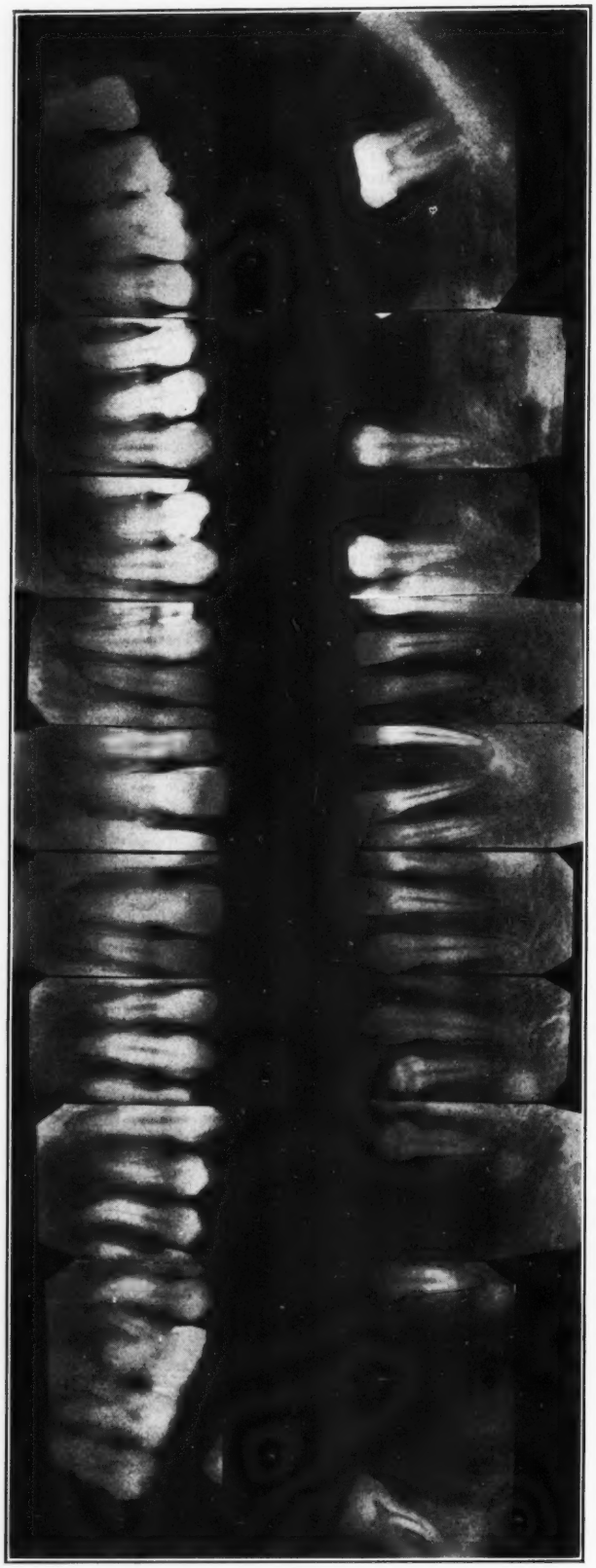

Fig. 40.—Muitiple root resorptions found together with hyperpituitarism. (Acromegaly.) No previous orthodontic treatment. Aged thirty-three years. (Patient No. 62.)

Fig. 41.—Multiple root resorptions found together with hyperpituitarism. (Acromegaly.) No previous orthodontic treatment. Aged twenty-five years. (Patient No. 86.)

Fig. 42.—Multiple root resorptions of maxillary and mandible teeth in a patient with hyperpituitarism. (Acromegaly.) No previous orthodontic treatment. Aged thirty-three years. (Patient No. 105.)

Fig. 43.—Multiple root resorptions found together with hyperpituitarism, following orthodontic treatment for one and one-half years. (Acromegaly.) Aged thirty-five years. (Patient No. 42.)

of bone formation and destruction in the medullary portion of the bone. As the result of this unbalance, osteoporotic lesions develop. These can be seen in the jaws as well as in other bones. From our standpoint it is important to know that these osteoporotic conditions are also accompanied by root resorptions. This is not limited to areas of decreased function, as one might imagine would occur in the anterior region due to growth, but are found throughout the mouth.

IV. SYSTEMIC DISTURBANCES OTHER THAN ENDOCRINE UNBALANCE

Among the systemic disturbances which did not fall into the various groups of endocrinopathies, several patients with acute and chronic infectious diseases were found. Intestinal disturbances were most frequent, and the possibility exists that disturbances in mineral metabolism resulted, leading to pathologic bone formation. It has to be kept in mind that any infectious disease can lead to an

Fig. 44. Fig. 45.

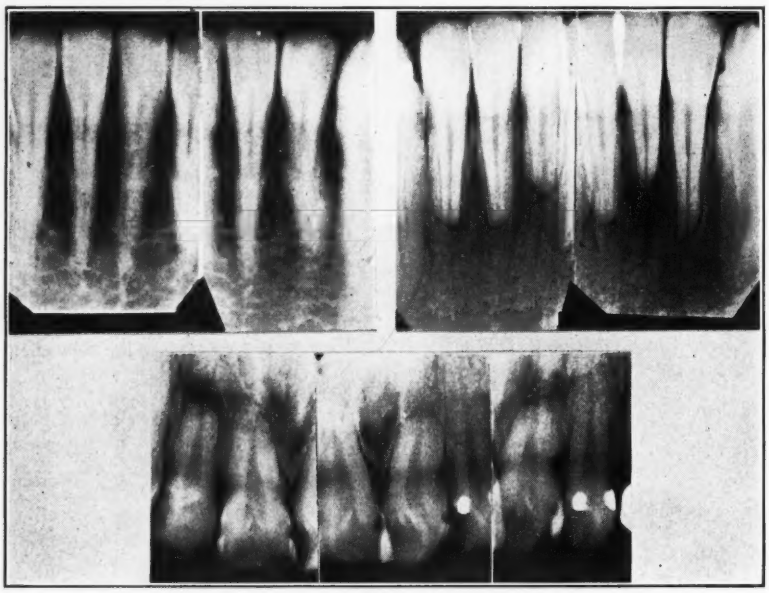

Fig. 46.

Fig. 44.—Root resorptions throughout mandible. Note marked horizontal atrophy of alveolar crest and severe osteoporosis. No previous orthodontic treatment. Aged forty-five years; systemic disease: duodenal ulcer for fourteen years with severe dietary deficiency. (Patient No. 35.)

Fig. 45.—Marked root resorptions following orthodontic treatment for ten years. Numerable severe infectious diseases during treatment. Aged eighteen years. (Patient No. 6.)

Fig. 46.—Multiple root resorptions in a young patient suffering from arthritis (?). No previous orthodontic treatment. Aged twenty-four years. (Patient No. 46.)

involvement of the osseous structure, either directly or indirectly, and until more data are available it seems best to advise the practicing orthodontist to exercise the greatest caution in the movement of teeth during the course of diseases accompanied by fever or in chronic diseases which have an influence on the circulatory system or on the general or mineral metabolism. (Figs. 44, 45, and 46.)

Finally, the central and lateral incisors of a patient suffering from hypothyroidism are shown in Fig. 47 before and in Fig. 48 after orthodontic treatment.

The brother of this patient, who had the same general disturbance, developed similar resorptions even though he had never been treated orthodontically (Figs. 49 and 50).

CONCLUSIONS

The question of the reason for root resorptions following orthodontic treatment has not been solved as yet. The statistical data just presented, as well as the clinical observations, have brought us a great step forward, but we are still far from being able to determine exactly the prognosis of treatment with regard to possible root resorptions. It is still not known whether a therapeutic dosage of thyroid extract will prevent root resorption in cases of hypothyroidism, or whether the treatment of any other endocrinopathies or systemic disturbances, before we place our appliances, will be of value in this respect. Experiments, however, along these lines are under way in my department.

Fig. 47. Fig. 48.

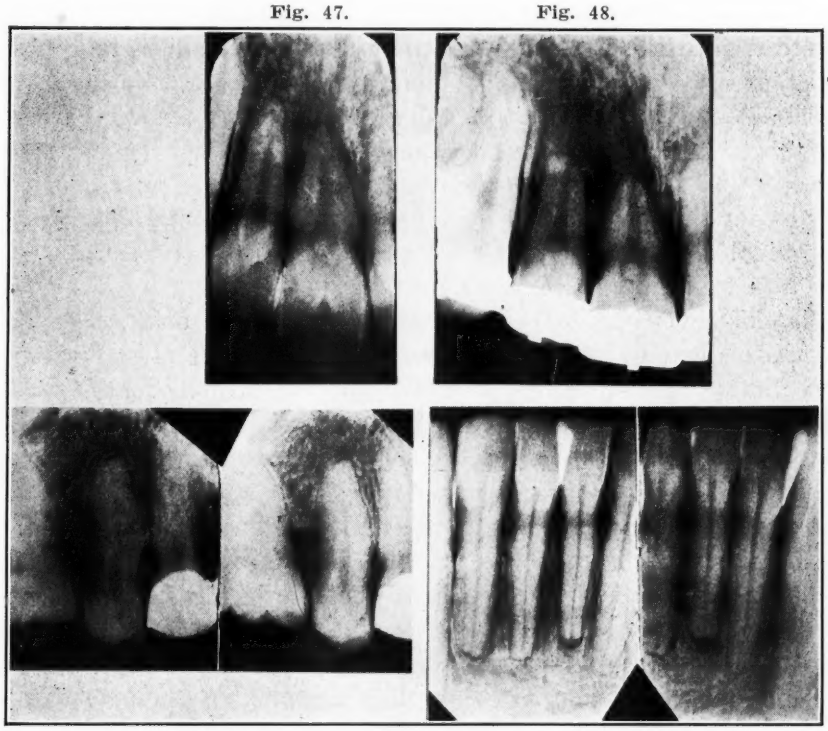

Fig. 49. Fig. 50.

Figs. 47 and 48.—Central and lateral incisors before and after orthodontic tooth movement. Aged nineteen years; systemic disturbance: hypothyroidism. (Patient No. 69.)

Figs. 49 and 50.—Multiple root resorptions found in brother of patient shown in Figs. 47 and 48, who had *not* been treated orthodontically but who had the same systemic disturbance: hypothyroidism. Aged twenty-eight years. (Patient No. 114.)

The limitations of these statistical data are obvious. The very fact that the reports which we have, have come from a large number of orthodontists makes it inevitable that many variables have entered into the investigation. That portion of the data which we obtained by definite tests and examinations in my department may perhaps be considered constant for this group, but that obtained from various orthodontists was at times incomplete and subject to variation.

We have followed in this investigation the definite policy of determining the frequency of systemic involvements in 100 cases of root resorption. The next phase of our work has to deal with the determination of the percentage of root resorptions in 100 patients who have systemic involvements of the types

noted in this report. In other words, we must reverse our program, take the opposite viewpoint and collect data of an entirely different nature. The results of this new project should make it possible to predict, with a fair degree of assurance, the percentage of root resorptions which will occur in patients suffering from any systemic disease at the time of orthodontic treatment.

In cooperation with the Orthodontic Department of the University of California, College of Dentistry, such an investigation was started August, 1934. A practical method has been outlined which can be followed by any orthodontist so that he may accumulate the data needed and thus be of service to the profession in bringing this work to a successful conclusion.

I hope within the next few years to be able to report to the profession additional data as to the chances of preventing root resorption in patients with given systemic disturbances. By that time, also, I should be in a position to state what percentage of over 200 patients now under treatment at the University show evidence of root resorption. While none of these patients show signs of this at the beginning of treatment, some of them have systemic disturbances. Thus they form a representative group such as might visit any orthodontist for the correction of dentofacial deformities, and these data should be most valuable in determining what percentage of root resorptions can be expected in a typical practice.

While this investigation has not given us the final answer as yet to the question, "How can we prevent root resorption?" it has already brought out one vitally important truth, that is, the institution of orthodontic treatment is by no means the sole factor in the production of the resorptive process of root ends so frequently observed during the course of treatment. Other factors, the etiologic force of which we are just beginning to appreciate, seem to have a tremendous importance in determining an individual susceptibility. One of the most important of these is undoubtedly pathologic bone formation.

Furthermore, it should be emphasized that the incidence of root resorption in both treated and untreated cases is a great deal higher than the profession realizes. It is incumbent, therefore, on the orthodontist to determine in any case presented for treatment, whether there are any of these factors present which, on the basis of this investigation, can be regarded as warning signals not only to be cautious in mechanical procedures, but also to take steps to control the biologic factors.

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DISCUSSION

Dr. Clay R. Murray, New York City.—I have been interested in the question of bone repair and bone pathology for some twenty years largely from the clinical standpoint, and rather extensively from the experimental standpoint.

My conception of bone formation is one which does not necessarily involve the activity of any cell type as a specific cell. As more information is accumulated, it becomes more and more apparent that it is entirely possible that the deposition of calcium in living connective tissue to form bone is a purely biochemical extracellular activity, the result of a local tissue death and local accumulation of calcium, a change in the biochemical status of the fluids and the redeposition of that calcium in the new tissue which replaces tissue death.

Whenever pressure is applied to bone. one of two things occurs. Either resorption occurs, which is commonly interpreted as death of bone, and probably erroneously so since it means merely decalcification of the structure that was called bone only when it contained calcium. On the other hand, if that pressure is not severe enough to produce fairly rapid tissue death, increased deposition of calcium occurs, and one obtains a concentration of calcium in the region of pressure rather than an absorption of calcium.

I know of no instance in clinical medicine where pressure on bone sufficient to cause rapid and marked absorption—mind you, I am speaking purely of pressure—can occur without the occurrence of symptoms to the patient in the form of pain and discomfort.

It is therefore with considerable surprise that, on reading Dr. Becks' paper, I found this extensive evidence of bone resorption in patients who presented no symptomatology as the result of the pressure phenomenon brought into play through the use of orthodontic sieges. I enjoyed reading Dr. Becks' paper, and I think I learned a great deal from it.

Among the products of civilization is apparently a deterioration of the entire endocrine system, which will call for something like a physiologic NRA if Dr. Becks' theories are correct. In order to bear out his findings, one would have to find that in one hundred patients, as they come into the clinic regardless of whether they have root resorption or not, less than 60 per cent showed evidence of hypothyroidism. One would also have to find out whether, in a group of patients who presented no evidence of hypothyroidism, less than 94 per cent have root resorption. If those correlating figures are obtainable and back up the findings which Dr. Becks has quoted in his paper, I believe the orthodontists can feel that from their standpoint there is no reason why orthodontia should be blamed for root resorptions.

One of the most interesting figures among those presented by Dr. Becks was the fact that in the cases treated, 24 per cent occurred in normal individuals; that is, the resorptions were in 24 per cent of normal individuals, and that on request for information from those who took care of these patients, 19 per cent had complained of pain during the course of their treatment. Evidently the percentage of resorptions in these normal individuals corresponded very accurately with the percentage of root resorptions accompanied by pain.

One other point which I should like to mention comes from Dr. Nicklas of Philadelphia and from Dr. Finkelstein of the Joint Disease Hospital in New York, who have been experimenting in recent years with preparing long bones in children for correction of deformity

by producing softening of the bone preliminary to correcting the deformity, and were unable to correct the deformity without surgical intervention. Dr. Nicklas produced the softening by dietetics, and Dr. Finkelstein by prolonged immobilization of the extremity, thereby causing decalcification of the bone.

Interested as I have been in the orthodontists' problems through the stimulating influence of Dr. Waugh, I thought possibly here lay a field in which investigation might make it possible not only to hasten the process of correction, but, so to speak, to prepare the field by first making the tissues more amenable to correction, and once the correction is secured return them to their normal.

Of course, the immobilization process of the human jaw would be entirely out of the question. The dietetic consideration seemed to provide considerable food for thought. However, after listening to Dr. Becks' presentation and the effect of the deficient diet on the jaws, on the teeth, and the roots of the teeth as regards resorption, I think there is still more food for reflection before any experimental work along this line is tried.

I shall look forward with interest to whatever Dr. Becks finds in his subsequent investigations, and I sincerely hope he will not leave me in ignorance of what he finds out. I shall be extremely interested to hear the results of his further investigations.

President Waugh.—This discussion will be continued by Dr. Irving H. Pardee, associate professor of neurology and chief of the Endocrine Clinic, Columbia Presbyterian Medical Center.

Dr. Irving H. Pardee, New York City.—I really have had practically no experience in orthodontia, except paying the bills for it. That is not nearly so delightful an experience as being here to talk to you.

As far as disturbances of the glands of internal secretion are concerned, I have had little more experience, and I am tremendously interested in the figures and the presentation which Dr. Becks has given. I had the privilege of reading his paper before he presented it.

In the first place, my personal reaction is that Dr. Becks has definitely established the fact that root resorption is not due to pressure. That must be an exceedingly important conclusion for all who are doing that work.

In the second place, I do not think he has definitely demonstrated what is the cause of that resorption. He knows that just as well as I do, because I talked with him beforehand.

In considering these cases, whose statistics he has shown you, we must, however, look upon them from the point of view of what kind of an individual needs orthodontic treatment. In other words, why should one person need it, and another one not? You might say that my child needs orthodontic treatment because I should have had orthodontic treatment, but that does not make any difference. The point is, why should my child need it? Why should I need it? Why should those who apply to you find it necessary to come for orthodontic treatment?

That depends upon the make-up of the individual. It depends on what his anthropologic make-up is, and his anthropologic make-up depends on certain factors; in the first place, his heredity and, in the second place, his internal glandular balance or imbalance, whatever that is.

Dr. Becks has shown cases that had root resorption in which the thyroid was deficient. He has shown cases in which the parathyroids were overactive, in which there was root resorption. He has shown 100 per cent of cases of acromegaly, which is a hyperpituitary condition, in which root resorption took place. Then his figures tell us that a large percentage of cases of root resorption are hypothyroid. That brings us to a question as to whether we have definitely established, or whether he has definitely established, that these cases are hypothyroid. Dr. Becks is just as alive to the question as I am, that the establishment of a diagnosis of hypothyroidism is difficult and cannot be made by means of the basal metabolic rate alone. The basal metabolic rate is not the only diagnostic factor in hypothyroidism.

In talking with Dr. Becks, he tells me that several men made examinations on these patients. In other words, it was the opinion of a number of different men as to whether the patients were hypothyroid or not.

Then, again, at least twenty cases out of forty in his Table IV, cases with orthodontic treatment, showed basal metabolic rates of minus 15, or higher. It is considered in certain laboratories that a basal metabolic rate between minus 15 and plus 15 is normal. In some laboratories. the figures are put at minus 10 to plus 10. So I think we have to reconsider those figures from that viewpoint. I am a little uncertain in my own feeling that all those cases were hypothyroid, as he reported.

When the thyroid gland is deficient, as we all know, there is failure of closure of the epiphysis, failure of lack of development. Therefore, does it produce resorption? We do not know that. We can presume that.

On the other hand, there are many cases of retarded growth that are caused by a pituitary gland disturbance. The anterior lobe of the pituitary gland secretes hormones into the blood which bring about growth. It does not bring about normal growth in the absence of thyroid, but it brings about normal growth in the presence of even a small amount of thyroid. The majority of dwarfs we see walking in the streets, in the circuses, and everywhere else, are undersize due to lack of pituitary stimulus.

There is another condition in the pituitary in which certain of the basophile cells will grow a tumor, or result in the same picture of decalcification of the bones as is seen in parathyroid disease. The parathyroid causes decalcification of bone. It causes cystic degeneration of the bone, and I wonder whether in many of these patients there was no particularly striking evidence of decalcification throughout the skeleton, but perhaps there was some disturbance of calcium metabolism. Perhaps there was some disturbance of vitamin D. These are important elements which have to do with the parathyroid glands.

The sex glands play some part in the maturing and growth of bone. The epiphyses in the bones do not close until the sex glands have completed their growth. This closure of epiphyses occurs around the time or shortly after puberty. This is the time when the sex glands reach their maturity.

Why is it that at that time we so frequently see such a marked and sudden decay of the teeth? Is this the age at which root resorption is more liable to take place, namely, between fourteen and eighteen years? I am asking you. I would think so.

So I should stress the fact that the thyroid gland, the parathyroid gland, the pituitary gland, and the sex glands all working together carry out and bring forth a normal and complete growth of bone and the maturing of the bony skeleton. Under those circumstances, we must not say it is entirely due to thyroid, because if we do there is going to be an awful lot of indiscriminate thyroid medication given, and if there is anything that is dangerous it is indiscriminate thyroid medication.

If the glands are a basic factor in root resorption, it is not one gland but many glands. It is purely a glandular condition. It is not only the thyroid, not only the parathyroid, not only the pituitary or the gonads; it is all of them somewhere failing to carry through properly their calcium metabolic function.

What need we do to see this problem through? In the first place, we must study very carefully in some of these patients their calcium metabolism and see why root resorption takes place. We ought to take a group of casual gland cases, hypothyroids, hyperpituitaries, hyperthyroids, gonad cases, sex cases of different groups, without any regard whatsoever to their teeth, and see in which of that group one will find root resorption.

Lastly, I think we should study more carefully the nutritional factors, the factor of vitamin D, of vitamin C, of the amount of calcium and minerals, which are present in the diets of these patients.

One of the most difficult studies is to determine what diet a person lives on, because the majority of people have no conception of what they eat. Answers which patients give you are entirely unsatisfactory, and for that reason that subject must be revamped.

I should like to congratulate Dr. Becks on bringing before you what seems to me really the meat of the matter, and that is that root resorption is not caused by pressure, but is caused by some metabolic, endocrine, or nutritional factor yet to be determined.

President Waugh.—It is said that orthodontics is both a science and an art. The effect of the two on groups of international relations seems to be very different among

ourselves as orthodontists when we discuss art with those of the foreign group. It seems to me it rather brings up international lines of difference. However, with the science pertaining to our calling, as with all science, it seems to transcend international differences and make brothers all one in science.

We have with us this afternoon a real scientist who, like Dr. Wingate Todd and Dr. Becks, has come to us from across the water and is contributing very largely to the progress of orthodontia.

Every rule in life, I presume, is made to be broken occasionally if broken wisely. I am taking the liberty of doing that this afternoon in calling unexpectedly on a visitor and a scientist whom you all know by his writings. I am going to surprise Dr. Rudolph Kronfeld, of Chicago, by asking him to step to the platform and talk on this subject for just a moment.

Dr. Rudolph Kronfeld, Chicago, Ill.—I have listened to Dr. Becks' paper, and I have seen his records and known about his investigations before I came to New York, but I made it a point to be here today and listen again, and I have listened to the very excellent discussions that followed this paper.

There is nothing I have to contribute, except to say that on the experimental end we have worked for many years in an attempt to produce root resorption in animals by mere gross and violent force, and have not succeeded. It is possible to produce shallow resorptions on the roots of practically all animals by force, no matter whether these animals are kept under sufficient or are kept on insufficient diet, and some of the resorptions Dr. Becks has shown today belong in this group. No resorption in animals I have ever seen, which was produced experimentally, compares with the razorlike cut through the root which is sometimes found in some of the human cases of the type that was shown today, and of the type I as a practitioner unfortunately have known in the clinic.

We have to be very careful in the application of experimental animal work. I think Dr. Becks has the right lead when he stresses the clinical investigation of the patients in the first place, and leaves the animal experiment, at least temporarily, alone, because the animal is apparently of a different constitution as far as the relation between tooth and bone goes.

Dr. Hermann Becks.—There are more than fifty questions I am supposed to answer, and I have been advised by President Waugh that the time is not sufficient to answer all of them.

In their absence, I want to thank Dr. Murray and also Dr. Pardee for their discussions. I think there is very little variation in our conception of bone formation and the relation between endocrine disturbances and bone formation.

I might emphasize also what Dr. Kronfeld has mentioned, that the forms produced in animals are not identical to the forms of root resorption we find in man. I might also emphasize that the resorptions we observe in orthodontic patients are much more marked. In other words, apparently the mechanical force accelerates the resorptive forces. I should also like to emphasize that I mentioned very definitely they are found in coexistence with endocrine disturbances and other disturbances. We should never make a contrary statement until we have definite data to prove it.

I should like to close the discussion by calling to your attention that individualization of each patient should be the outstanding feature of your practice in the future. In other words, to individualize in each case what the biologic factor is.

I mentioned that there might be many representative groups of disturbances; let us say, endocrine disturbances, constitutional diseases, dietary deficiencies, and perhaps even age might play a rôle, or other factors. If we take typical representatives of each group, throw them all in one box and shake them well, and then take out five and say, "All right, we will give you cod liver oil and stop root resorption," that would not work. In other words, we shall have to come to the determination of biologic factors involved in each patient, which will require an entirely different curriculum for dental or orthodontic education from that which we have at the present.

President Waugh.—For the comfort of those who have asked these questions, it is thought best not to have them discussed this afternoon because the most interested members of the attendance will have the opportunity of hearing Dr. Becks tomorrow afternoon in those one and one-half hour educational clinic classes that are being conducted. If those classes are not as yet entirely filled, the members and guests may have the privilege of spending one and one-half hours with Dr. Becks in one of those clinics tomorrow afternoon.

Also, may I ask that everybody step into the Astor Gallery, into the scientific room, and study there the large number of radiograms which Dr. Becks has on exhibit in corroboration of the stand he has taken in the presentation of his paper. We are deferring the time so that Dr. Becks may have better opportunity to answer these questions fully.

GROWTH OF THE JAWS AND THE ETIOLOGY OF MALOCCLUSION

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(*Continued from page 368, April*)

CHAPTER IX

ETIOLOGY OF MALOCCLUSION

In the preceding chapter it was shown that when the normal processes of growth are interfered with, malformations of the jaws and malocclusions of the teeth may develop. The interference was considered to be of a general character, and according to Jansen's theories the degree of the resulting deformity is in direct proportion to the intensity of interference. In the study of the causative factors it must be considered that malocclusions may be caused by local as well as general injurious influences. The general causes on account of their nature interfere with the physiologic growth processes, while the local causes disturb the physical forces which must necessarily be present during normal development.

The general causes may become operative before or after birth, and the earlier in life they appear, the more likely growth will be affected. Severe deformities invariably originate in utero as a result of a general infection or debility of the mother, so that at birth the extent of the future abnormality is already determined; which may be evident at that time or appear in later life. Thus it is clear that all individuals do not start postnatal life under the same conditions. Even under identical environmental conditions the development of each individual will be different, which is determined partly by conditions impressed upon the individual before birth and partly by inheritance. A fine distinction must be drawn between inherited characteristics and characteristic deformities caused by injurious influences before and after birth. Inherited characteristics may or may not produce a normal individual, so that even under the best of conditions a deformity may occur in a child. It frequently happens that on account of complex growth processes an otherwise normal child shows a deviation in the development of the dental apparatus which can be classed as a deformity. The perfect health of the mother during pregnancy and the uneventful history of the child exclude the possibility of nocive influences in such instances, and another explanation of the causes of the deformity becomes necessary.

In order to understand better the etiology of such deformities, let us consider any number of normal individuals. Even on a casual examination we differentiate between the two sexes; we observe differences in height, features, hands, feet, etc.; yet there is something about all these individuals that makes them normal. A more detailed study of the measurements of parts, and relative

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measurements of parts, will show a great variability, which in the final analysis accounts for the differences observed on a superficial examination. We further come to the conclusion that the relationship of the parts and their relative measurements determine normality. Measurements alone will not define normality, and positional relationship must always be taken into account. It follows, therefore, that for normal development normal positional relationship must be maintained during growth, which is possible only if the relative rates of growth in the same direction of the related parts are also maintained. This does not imply that the relative rates of growth in any direction in different individuals are the same, but that the rates of increase in any particular direction of related parts of the same individual must be in harmony.

We too often observe great resemblance between members of the same family. A resemblance means similarity, and similarity can be accounted for only in terms of proportionality, which in turn establishes definite ratios between the measurements of the parts. These ratios are the relative measurements of the parts, and they can be maintained during development only if the relative rates of growth persist. Family resemblance may be a resemblance of normality, but often that of abnormality, so that similar abnormalities may be present in each of the several members of a family. These abnormalities are inherited and are not caused by injurious agents during growth. In the final analysis, however, the abnormality is only a manifestation of a disharmony in the relative rates of growth, and it would be more proper to speak of "inherited disharmony in the relative rates of growth" than of inherited abnormality.

A distinction must be made between an abnormality established by inheritance and one resulting from a feebleness of growth induced by an injurious agent. From an orthodontic point of view the former represents a condition in which bone growth is normal, and the abnormality is not the result of an interference with the growth processes. In the latter, growth is interfered with, and the reaction of bone to pressure stimuli must be different than that of normal bone. This difference is important. Jansen pointed out that bone affected by feebleness of growth responds more readily to pressure than does normal bone. In orthodontia this is confirmed by the ready response to treatment of some cases which are definitely affected by feebleness of growth. On the other hand, it is most difficult to correct conditions of the inherited type. This is contrary to the belief that individuals having the best development are the best subjects for orthodontic treatment. It is to be remembered, however, that growth is an unfolding process and at some period a point is reached beyond which appreciable growth cannot take place. A part may be developed to its utmost limit by use and exercise, but when that part reaches its full development further efforts will lead only to injury. Similarly, if a malocclusion is the result of a pure inheritance, it is more difficult, if not impossible at times, to bring about sufficient development to establish permanent normal relationship. Such cases if corrected are stretched beyond their normal limits of growth, and they are more likely to resume their earlier form than cases affected by feebleness of growth which have not reached their full development. This is further substantiated by the fact that a muscle or bone subjected to heavy use develops to cope with the increased demand, but as soon as that demand on the tissues becomes normal, the parts return to their normal condition.

Thus a malocclusion may be inherited or acquired. But, to generalize, it must further be recognized that normal occlusion is also due to inheritance, so that every individual inherits certain relative rates of growth which may or may not tend toward normality. When the relative rates of growth are in harmony, the individual will develop normally; when in disharmony, the development of the individual will be affected accordingly.

The acquired type of malocclusion is always caused by noeive influences or injuries, before or after birth. These influences have an effect on the growth processes, and the relative rates of growth are altered. Thus an inherited malocclusion may take on a more severe form under these influences, and the potential normal occlusion may also develop into a malocclusion. Since it is universally recognized that malocclusions may be acquired or inherited, theoretically there are three different kinds of malocclusions:

1. Malocclusions due to pure inheritance.
2. Malocclusions due to injurious influences operating on a potential normal occlusion.
3. Malocclusions due to injurious influences operating on an inherited malocclusion.

From the orthodontist's point of view this classification is of practical importance. It includes all possible causative factors and segregates malocclusions according to their probable response to treatment. Malocclusions due to pure inheritance are difficult to treat, while those due to injurious influences operating on a potential normal occlusion present the most favorable conditions.

This classification of malocclusions may have a far-reaching effect on the further development of orthodontia. The etiologic factors if recognized will have a new meaning in diagnosis and will point the way to a clear-cut decision in regard to prognosis. The probable response to treatment is an important consideration in every instance, and the proper evaluation of the effects of the several etiologic influences will enable us to predict with more or less exactness the probable outcome of a case. The classification is theoretical and is based on the postulated propositions that every individual begins life with an inheritance which determines the relative rates of growth during normal unhindered development, and that these relative rates of growth may be altered by injurious influences. Some authorities exclude heredity from the etiologic factors and look upon malocclusions as the result of local and environmental conditions. A closer analysis of such views, however, will only confirm that inheritance must play a part, for if it is accepted that local and environmental conditions are capable of bringing about malocclusions, the implication is that the development of every individual will proceed normally unless subjected to these influences. According to this conception, malocclusion cannot be inherited. Even if it is granted that this is true, the assumed universal tendency toward normality must be interpreted as an inherited tendency. These differences of opinion arise as a result of misunderstanding. If inheritance is defined as the transmission to the child of a condition which existed in a parent, then the exclusion of heredity from the etiologic factors is justified. Under this definition pure inheritance

is not possible, for the ovum and the spermatozoon are both potential carriers of the hereditary factor, and an inherited condition may originate in either and be modified by the other. The inheritance may be defined on a much broader basis. It is a well-known fact that living organisms reproduce their kind, and as Wallace states: "The fertilized egg cell implicitly contains in some way which we cannot imagine, the potentiality of a living creature—a tree, a daisy, a horse, a man." Now these potential living creatures develop into the likeness of their parents, so that the offspring of a tree is another tree, that of a horse is another horse, etc. The development of the offspring proceeds according to the characteristics contained in the fertilized ovum, and the characteristics responsible for the reproduction of the *kind* are inherited. The resemblance of a child to its parents is brought about by a more specific inheritance. Within the same species, generally, there is a closer resemblance of the offspring to the parent than to other members of the same species. But since we have already shown that resemblance means similarity, and similarity can be expressed only in terms of proportionality, we must conclude that the relative rates of growth of the future individual are already determined in the fertilized egg cell, and for this reason they are inherited. Thus every individual begins life with an inheritance, which does not necessarily mean that the abnormalities of the parent will be transmitted to the offspring, but that *the offspring is endowed by the parents with certain growth characteristics, which are entirely individual.*

With this understanding of inheritance we may have an inherited malocclusion which does not exist in the parents, and such conditions may be brought about by the modifying influence of one parent cell upon the other. If we admit the possibility that malocclusions may be produced by local and environmental conditions, we must also admit that they may develop from inheritance, for the assumption that growth and development will proceed normally unless interfered with carries with it the exclusion of another possibility which is not permissible. Thus, from a theoretical point of view, an individual may begin life with an inherited tendency to normality or with an inherited tendency to abnormality. During development these tendencies may be modified by injurious influences, so that under all conditions of inheritance an abnormality may develop. This is the basis of the classification, which also differentiates between growth processes and development.

In an inherited tendency to normality, the growth processes are normal and development proceeds normally. In an inherited tendency to abnormality, the growth processes are also normal but the development is abnormal. If an abnormality is the result of injurious influences, both the growth processes and the development are abnormal. This will throw further light on the possible reaction of bone to orthodontic treatment. In all inherited conditions the growth processes are normal, so that the reaction of bone to pressure stimuli will take place according to Jansen's pressure-growth curve for the normal individual. In these cases the response is slower, and tooth movement is more difficult. Acquired abnormalities are characterized by an interference with the growth processes, and the reaction of bone to pressure stimuli will take place according to Jansen's pressure-growth curves for individuals affected by the different de-

grees of feebleness of growth, provided that the injury is of a general character. These cases respond more readily, and tooth movement is accomplished with less difficulty.

When the growth processes are affected, the development of the individual is also affected. If we understand that development is the unfolding of the individual and the establishment of normal relationships between its parts, it will be seen that development may proceed unhindered to a certain point beyond which only slight changes will take place. This point represents the maximum development under normal conditions and is determined by inheritance. Under mechanical stimulation the dimensions of a part which attained full normal development may be changed, but as soon as the mechanical stimulation ceases, the part slowly resumes its former dimensions. In other words, *an alteration of the form predetermined by inheritance cannot persist.* The development predetermined by inheritance may be normal or abnormal, so that *a corrected inherited abnormality cannot persist;* thus, a part cannot be changed permanently beyond its predetermined limits.

In the ultimate analysis, predetermined development is identical with the predetermined rates of growth, and relative rates of growth, and conclusions drawn from the effects of injurious influences on the rates of growth will also apply to development.

With these conditions in mind the study of the etiology of malocclusions takes on an added importance. Heretofore, the knowledge of the exact cause of a given malocclusion aided only in the removal of some local influences, and no predictions could be made in regard to the possible response to treatment and to the probable termination. With our points of view, the determination of the etiology of a case takes on the form of diagnosis, from which valuable advance information may be obtained. In order to be able to differentiate between inherited and acquired causes of a given malocclusion a complete history of the patient becomes a necessity. This history must be so arranged that the various diagnostic factors stand out in proper relation to each other. So far, the local causes have not been considered, for they form only a supplementary group to the more general injurious influences. The local causes are very important, for they are often responsible for malpositions of teeth extremely difficult to correct, and for this reason they will be taken up separately.

The diagnosis of a case begins with a survey of the general conditions present. The relationship between the maxillary and the mandibular teeth should be noted, which is an index of the degree of jaw development. In general, the Angle classification serves as a sufficiently reliable guide. Too much reliance should not be placed on the classification, and in many instances a more accurate analysis of jaw relationship becomes necessary. The next important step is to obtain the patient's history; this may be divided into three main parts:

The first division contains all data which are obtainable directly from the parents. A malocclusion may be transmitted to a child by either one of his parents. In cases in which there is a similarity of malocclusions between the parent and the child, we are dealing either with a malocclusion due to pure inheritance, or with a malocclusion due to injurious influences operating on an inherited malocclusion. There is a remote possibility in such instances that the

malocclusion is due to injurious influences operating on a potential normal occlusion, but from the data contained in this division of the history definite conclusions cannot be drawn. The probability is, however, that the malocclusion is partly inherited.

The second division is the prenatal history. As we have seen, the condition of the mother during pregnancy may be the cause of a malocclusion in the child. For this reason the condition of the mother during pregnancy should be carefully studied. In this connection it is important to know:

1. How many children were born before the patient.
2. If mother worked during pregnancy (home or business).
3. Social duties.
4. Fatigue.
5. Disease.
6. Nutrition.
7. Constipation.
8. Vomiting.
9. And any other information.

This information is necessary in order to determine whether the fetus was subjected to the injurious influences of nutritive, toxic, or infectious agents. Our present knowledge does not permit us to draw the conclusion that prenatal influences affect growth in the same order as determined by the table of relative rates of growth. It is certain, however, that for the severe types of malocclusions the foundation is laid at this period and in very early infancy. A knowledge of the relative rates of growth of the face of the fetus throughout the entire prenatal period would enable us to state with greater exactness the probable effect on development.

If severe disturbances affected the mother during pregnancy, the malocclusion is at least partly acquired, and it should always be remembered that due to inheritance the tendency of growth may be toward normality or abnormality.

The third division is the postnatal history, which includes the periods of infancy and childhood. We have already studied the effects of nocive influences on growth and the probable development of malocclusions, which, according to application of Jansen's theories to the table of relative rates of growth obtained from Hellman's measurements, takes place in a definite order. The age at which the injurious agents become operative is of the utmost importance, and their intensity should also be noted. Parents usually give reliable information as to the intensity of a past disease, and their impression as to the severity of the illness is sufficient.

According to the time of occurrence and the intensity of the disease, the effect on growth may be mild or severe. The earlier in life it occurs, the greater will be the effect; but, if a case is still normal when the permanent first molars establish their occlusal relations, in all probability the dentition will be completed normally. With a history of early severe diseases of childhood, the malocclusion is again at least partly acquired.

In order to differentiate further between inherited and acquired malocclusions, the effect of these diseases on the musculature and on the calcification of bone should be studied. Jansen enumerates four characteristic muscular symptoms of the feebleness of growth:

1. Flat feet.
2. Protuberant abdomen.
3. Round back.
4. Blue and moist hands.

These symptoms indicate that the musculature is too feebly developed to bear the body weight, to resist abdominal pressure, to balance the respiratory forces, and to resist hemostatic pressure properly.

Since according to Jansen's theory the musculature is always affected before the skeleton, some of these symptoms must be present in all cases of malocclusions acquired through general injurious agents. If these are not present, the injury occasioned by the disease was not of sufficient intensity to affect the growth of bone, and the malocclusion must be looked upon as inherited.

A further check upon acquired malocclusion may be obtained from the studies of Clinton Howard on the calcification of the bones of the hand and the wrist. According to this investigation, the twenty-nine centers of calcification begin to calcify at successive periods in regular order. From the number of calcified centers present at any age, the retardation of the growth processes may easily be judged. Boys are one year behind girls in the appearance of the centers of calcification.

While it is impossible at present to determine exactly whether a malocclusion is due to pure inheritance or to noxious influences, we can come to a fairly accurate conclusion as to whether a malocclusion is due mostly to inheritance or to injurious agents. And from this, the probable response to treatment and the permanency of the result can be predicted.

(To be continued.)

PREVENTION OF DENTAL CARIES IN CHILDREN*

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DENTAL caries is the child's cost of civilization. It is rare among primitive people and is rampant upon contact with civilization. It rises during growth and sets with maturity. It advances with refined living and is not arrested by any one of the current methods—dental or medical.

Dental decay is wide spread among all peoples—urban and rural; among all classes—rich and poor; among all bodily states—general good health and systemic disease.

Its universality has thus far defied the recognition of any one determining factor that is productive of dental decay. Dental studies of several centuries have been needlessly confined to teeth as entities distinct from the body that bears them, and so have their results been futile in arresting the progress of the most prevalent disease in civilized communities.

The tooth may be an end-organ, but it still has vascular and lymphatic relationship, even to the dentin, with the rest of the organism so that its life course cannot be determined by local conditions alone.

Systemic studies have more recently stressed tooth development more particularly from the standpoint of the newer knowledge of nutrition with due credence to the formation of the anlage or to the dental environment in caries. Teeth are in the first line of defense in the transition between environmental offense and systemic resistance, but they are slow to yield to disturbance in comparison with other tissues of the body, deciduous teeth reflecting damage three years after its onset and permanent teeth over six years after dental disturbance. Studies on dental decay must necessarily invite both dental and medical cooperation simultaneously in both clinical and experimental evaluation of the factors operative in their destruction.

Dental caries is a syndrome of many systemic and local disturbances rather than a simple disease of single cause. Every advocate of any one local or systemic factor as determining the nature of the process has few clinical adherents.

Our study of the relative values of acid- and base-forming diets upon the metabolism of rats has very definitely demonstrated that a preponderance of alkali-forming minerals are indispensable in superior growth and development as well as in the prevention of rickets. Similar observations were made on infants and children convalescing from various disturbances. We have since been able to accelerate the convalescence and buoy up the developmental progress of undernourished children by the daily administration of base-forming dietaries abundant in milk, fruit, and vegetables to the extent that each

*Presented at the meeting of the Section on Dentistry for children of the Second District Dental Society of the State of New York, November 25, 1935.

meal was definitely base-forming in content. Such formulation of the child's dietary from its total acid-base content confirms very definitely the actual composition of the child's tissues which during growth are ever excessive in alkali-forming elements.

Continuous administration of such a dietary regime accelerates the child's rate of growth, its development, metabolism, and activity in striking contrast with children maintained on acid-forming dietaries. It was incidental to these early studies that arrest of caries was observed in children maintained on base-forming dietaries. But it was not until a raw base-forming dietary was instituted in the study of anorexia in certain children that more striking arrest of dental caries became apparent in repeated routine examinations of these children. The metabolic significance of the base-forming dietary in dental development and the raw factor of foods in their relation to primitive dietaries that are protective of dental caries lead us to a more extended study of the relationship. Our qualitative observation of the number of carious teeth gave way to the quantitative calculation of the index of dental caries devised by Bödecker.

Heretofore an estimate of the degree of dental destruction in man from caries has been made only roughly. As long as the prevention of this disease was regarded as being possible solely through oral hygiene, exact statistics on the activity and distribution of dental caries were not considered necessary. Now, however, with more and more evidence indicating that some systemic factor or factors may augment or retard the activity of dental caries, it is essential to obtain exact data.

Evaluation of the degree of dental destruction should become part of the routine practice of clinical dentistry. A great number of cases is necessary to ascertain the average degree of dental destruction present in children and in adults. For this end a uniform type of tabulation, or caries index, is required.

The teeth of human beings have an average of five areas, of varying susceptibility to dental caries, making a total of 160 areas for the 32 teeth. In order to estimate the percentage of tooth surface destruction, a table of figures was set up giving a 100 per cent dental caries index when all of the 160 areas are affected by decay. Eighty affected areas give a 50 per cent caries index; one area gives an index of 0.625 per cent.

No table of figures is needed to compute the caries index of the deciduous, or temporary, teeth. These, being twenty in number, each having five surfaces, make a total of one hundred surfaces. Thus the number of involved areas of the deciduous teeth gives a figure expressed on a percentual basis (16 involved surfaces—16 per cent caries index).

A sketch of the mandibular left posterior teeth shows that each tooth may have five to six caries susceptible areas. Assume that in a caries ravaged mouth, twenty-four of these areas have been filled; this would indicate a caries index for these five teeth alone of 15 per cent. The number of fillings, however, would not necessarily be twenty-four, because often one filling involves two or even three surfaces of the teeth. In order to calculate the caries index, the number of areas involved should be counted and not the number

of fillings. The fillings here indicated are comparatively small, a condition that exists only in patients who have had constant dental care. If caries has been rampant, these teeth would show even less tooth tissue than indicated and more filling material.

To simplify the charting of the teeth and to ascertain quickly the caries index of each person, a diagram is suggested. This shows the thirty-two permanent teeth at the left, and the twenty deciduous, or temporary, teeth at the right. This form is used to chart the dental conditions of school children by dental hygiene students. Its purpose is principally to calculate caries indices of large groups. If such charts were to be used in conjunction with hospital records, the question on health would naturally be omitted.

Two separate caries indices must be set up, one for the deciduous and one for the permanent teeth. Otherwise a child of six years, having a high index as a result of a severe involvement of the deciduous teeth, would have a lower index at fourteen years of age when these teeth are lost. This is, of course, an impossibility, for a decayed tooth cannot again become perfect.

The distribution of a preliminary study of one hundred cases showed that children with a life caries index of 0 per cent were of the more stable elements of their race. They were all provided with base-forming dietaries containing an abundance of raw food. Their mouth hygiene was particularly noteworthy in comparison with two other groups of children revealing high indices of dental caries.

Studies of dental caries in various races revealed that those living in uncivilized or natural conditions are relatively free from caries in comparison with modern races whose percentage runs as high as 95 per cent. In fact, the number of teeth affected with caries in each individual studied was far less in those leading natural lives than in those under the influence of refined civilization, the ratio being 2 per cent in the former to 50 per cent in the latter.

THE DEVELOPMENT OF DENTAL CARIES WITH AGE

AGE (YEARS)	LIFE CARIES INDEX (AVERAGE FOR AGE)		NO. CASES
	DECIDUOUS	PERMANENT	
4	3	----	13
5	15	----	11
6	18	0.75	15
7	10	2.77	6
8	15	3.02	12
9	12	4.04	8
10	10	3.77	15
11	3	5.15	9
12	7	5.32	6
13	5	5.12	2

Finally it has been observed that the Maori race, subject to the influences of civilization for about one hundred years at the very most, has shown a rise to 93 per cent in its incidence of dental caries although the extent of caries in each mouth is comparatively low. Civilization per se is not so much responsible for the difference in the extent of caries but more particularly the nature of the civilized dietary which the particular race has attained. Mouth

hygiene was attained in no small degree even in uncivilized individuals by virtue of the hard usage of the teeth and jaws exercised in slow serious mastication to the point of being worn.

The incidence of caries increases rapidly with the age in childhood. The evaluation of the life caries index for both deciduous and permanent teeth at each age level from four to fourteen years reveals this upward trend in dental decay. The relatively low caries index for the deciduous teeth at four years becomes markedly increased at five years and reaches its peak at six years. Then the relatively low life caries index for the permanent teeth steadily rises until its peak at pubescence. With this increase in caries of the permanent dentition there is, of course, a slow decrease in the decay of the deciduous teeth. Not only does the number of carious teeth increase, but there is evidently a more deep-seated involvement of the disease in each tooth examined.

DESCENT AND DEGREE OF DENTAL CARIES

LIFE CARIES INDEX 0%	NO. CASES	L. C. I. 0-15%	NO. CASES	L. C. I. 15%	NO. CASES
American	6	Italian	30	Italian	16
Porto Rican	3	American	9	Porto Rican	6
Italian	3	Porto Rican	5	American	4
Portuguese	1	Irish	4	German	3
		Scotch	1	West Indian	2
		French	1	Czecho-Slovakian	1
		Russian	1	Finnish	1
		German	1	Greek	1
				Lithuanian	1
	13		52		35

Caries of the deciduous teeth is extensive and too prevalent to have been ignored in the periodic supervision of early childhood. The age range of the children under our observation has been up to fourteen. Their involvement in dental caries is not nearly so extensive as that reported for adolescence. Our observations reveal a peak of caries for deciduous teeth at six years and another peak of caries for the permanent teeth at twelve years. Other observers have recorded a peak of caries for adolescence at twenty years. This rhythm in the incidence of caries parallels that of development, thus relating both to deep-seated metabolic changes in the growing body.

The great prevalence of mouth and upper respiratory infections in children parallels somewhat the extensiveness of dental caries during growth and development. Both offenses abate with maturity. A study of the various factors involved in the development of dental caries, the relative occurrence of various types of such infections, was determined for a possible relationship to either the production or the maintenance of dental caries.

Two of the group with a light caries index of zero were hemophiliacs under my observation for several years. One showed an extensive chronic sinusitis with periodic cervical lymphadenitis, and the other boy showed markedly hypertrophied and diseased tonsils meeting in the midline and yet neither chronicity of infection proximating the teeth affected them in the least. The hemophilia in these boys prevented surgical clearance of these

infections, incidentally giving us an indication of their possible relation to dental decay. The associated acidosis with each of the infections which befell the children with no dental caries was necessarily transient enough not to make any lasting destructive effects upon the teeth. Our attempts to relate any type of infection in the region of the teeth with their possible destruction have led to no positive correlation.

ANALYSIS OF DIETARIES OF RACES IMMUNE TO CARIES

RACE	DAILY FOODS	DIETARY PROPERTIES
Maori	Kumara (sweet potato), raranhe (fern-root), taro (vegetable), bread (bulrush pollen and hinantree berries), hue' (pumpkin), berries, jellies, (seaweeds), fish and birds (delicacies)	Masticatory, raw and steamed, variety, alkali-forming
Eskimo	Fish, seal, walrus, white whale, birds, berries, reindeer moss, plants (20 kinds)	Raw, masticatory, acid-forming
Lapp	Milk, sorrel, butterwort (plants)	Raw and steamed, masticatory, alkali-forming
Indian	Fish, roots, grasses, fruits, nuts, berries, maize, potatoes, sugar-pine	Raw, masticatory, alkali-forming
Fuegian	Fish, dried fungus (mushroom)	
Fijian	Fish, opossum, pig, yams, taro, bananas, cocoanuts, sugar-cane	Raw and steamed, masticatory, acid-forming
Papuan	Kumara, sago, beans, game, cabbage leaves	Raw and steamed, masticatory, alkali-forming
Malay	Yams, roots, fruits, rice, maize, bananas	Raw and steamed, masticatory, alkali-forming
Jakun	Fruits, rice, kulim leaves (onions), wild ginger, tapioca	
Hebrides	Fish, liver, oatmeal, eggs	Raw and cooked, masticatory, acid-forming

Aciduric and acidogenic bacteria have been found in active carious cavities favoring the fermentation of carbohydrate to lactic acid. Their acid formation gradually dissolves enamel under the films or plaques covering the teeth. The *Bacillus acidophilus* in its various forms in the mouths of susceptible and immune persons, respectively, is, according to Bunting, the most consistent difference observed between these two groups. While we have studied the oral flora of children with and without caries, we have observed no differences in their teeth so far as all other types of infectious involvement about the teeth are concerned—fusospirillary infections, streptococcus tonsillitis, tuberculous adenitis, chronic sinusitis, pharyngeal abscess formations, gingivitis, acute contagious diseases, etc.

The nature of the infant's dietary affects the incidence of dental caries. A study of the children's dietaries during the first year of life in relation to those who were caries free as well as to those who had extreme caries showed no single nutrient as the determining factor either in the prevention or in the

production of dental caries. Most of the children observed have had the infant dietaries supervised in the same institution, and so available records were used in these comparative studies.

DEGREE OF DENTAL CARIES AND NATURE OF INFANT NUTRITION

LIFE CARIES INDEX	DURATION BREAST FEEDING				VITAMIN SUPPLEMENT				MINERAL CONTENT	
	0	1-3 MO.	3-6 MO.	6-12 MO.	O	C	D	C & D	ALKALINE	ACID
0%	1	2	3	8	2	6	–	6	10	3
0-15	2	8	4	3	11	14	1	13	18	26
15-	6	5	4	2	12	13	–	10	6	30

Breast-fed infants showed no greater freedom from caries than those artificially fed. In fact, there appeared no relationship between the duration of nursing and the development of caries.

Children free from caries were either artificially fed or breast fed, and children with extensive dental decay showed similar distribution in the type of early feeding. Even prolonged breast feeding far into the second year of life in some of the Italian infants produced no immunity to caries. In this respect severe dental caries in exclusively breast-fed infants was about as prevalent as severe rickets or scurvy in completely breast-fed infants.

The nutrition of the teeth is apparently better conserved during the prenatal than during the postnatal life, but breast milk does not contribute to the dental structure such a degree of nutritional protection. Just as breast milk of healthy mothers can be modified by antirachitic measures in the mother's dietary for the breast milk to be antirachitic to the infant, so will it likewise be possible to modify the breast milk for its protective effect upon dental caries to the infant.

Breast milk is nonprotective of dental caries as has previously been recorded by other observers. In a survey of 15,000 school children in England, Pedley found that caries was present in breast-fed children almost as much as in the artificially fed. Jones found that 85 per cent of Japanese infants in Hawaii showed a marked destruction of deciduous teeth in spite of the fact that 90 per cent of them had been breast fed. Careful and periodic supervision of infants and children in the course of an active pediatric practice likewise reinforces this observation. We cannot assume that breast milk can contain destructive constituents in so large a racial group, but rather that some nutrient factor was inadequate for the maintenance of dental metabolism.

Development of the teeth begins in intrauterine life with deposition of lime salts in deciduous teeth at about the sixteenth week. The nutrition of the mother hardly influences the teeth of the fetus because embryonic tissue possesses considerable growth impulse enabling it to remove the necessary growth constituents from the maternal structure. Even if the maternal diet be inadequate, the mother is more likely to be affected than the fetus, unless her dietary be extremely deficient for a prolonged period, when both mother and fetus necessarily suffer.

The maternal organism can supply all nutrients but vitamins A, C and D which are not stored in sufficiency. It is only during nursing that the deficiency in these constituents is markedly reflected in the composition of the breast milk. If these vitamins were the determining factors, the structure of the permanent teeth might be involved. Thus far clinical evidence is lacking that these vitamins necessarily dominate dental development in infancy.

Degree of Dental Caries and Incidence of Oral Infection

LIFE CARIES INDEX	ACUTE			CHRONIC		
	NONE	MILD	SEVERE	NONE	MILD	SEVERE
0%	1	2	3	1	3	3
0-15	7	16	6	7	14	9
15-	4	4	6	4	11	10

The children found free from caries received no greater vitamin supplements than did those with marked caries. The distribution of those who received adequate intake of vitamin C as well as D is about the same in each of the three groups of nil, moderate, and extreme caries. Additions of orange juice or cod liver oil appear to be unrelated to the degree of dental caries in each of these groups. Children with rickets in infancy showed no more marked degree of caries than those who were free from rickets. In fact, a number of the mothers of those infants who showed dental caries received adequate doses of cod liver oil during the last two to three months of pregnancy as part of their prenatal supervision. Two of the fourteen children free from caries had both clinical and chemical evidence of rickets in infancy, while relatively larger numbers of those who had dental caries were clinically rachitic in infancy. From our careful observations of one hundred cases studied for dental caries, 20 per cent had evidences of rickets in infancy, but no relationship can be established between the occurrence of rickets and the subsequent development of dental caries.

The children whose life caries index was zero were all maintained on a base-forming dietary in infancy. In addition to the breast or artificial formula feeding, they were offered beginning with the first months of life daily additions of stewed fruits, fruit juices and banana, vegetable purée and potato, two to four times daily and in addition a single portion of acid-forming cereal. These children were among the first to have been given the advantages of early semisolid feeding in the first months of infancy primarily for the maintenance of an alkali-forming regime consistent with calculated mineral requirements during growth. Four years ago, I summarized work that had been in progress since 1926, that standard infant dietaries were acid-forming, contrary to the composition of the child's body, and contrary to requirements in each of the mineral constituents essential for growth. It was on this basis that early semisolid feeding was instituted in the first months of life to bring mere milk and carbohydrate intake to the requisite levels of mineral requirement.

Our studies of the infant dietaries of the children with no caries reveal the base-forming factor in the dietary as the predominant one in comparison with the two other groups of moderate and extreme caries who were main-

tained in infancy on a more or less neutral or acid-forming regime. While no single dietary constituent appears as the determining factor so far as infant feeding is concerned in the evaluation of dental caries, the nature of the total mineral intake, acid- or alkali-forming, parallels the incidence of dental caries. Comparable to these observations appears the work of Jones indicating arrest of dental decay in Hawaiian infants after six months of constant administration of fruits and vegetables in addition to milk.

DENTAL CARIES AND KETOSIS

CASE	AGE	DESCENT	LIFE CARIES INDEX DECIDUOUS	PERMANENT	DURATION DIET
		I. Acid-Forming Ketogenic Diets			
H. M.	10 yr.	German	19	5.0	6 mo.
H. L.	10 yr.	German	7	5.0	8 mo.
L. P.	7 yr.	French	17	5.0	1 yr.
R. V.	9 yr.	American	25	5.0	18 mo.
J. K.	8 yr.	American	23	3.7	1 yr.
J. M.	10 yr.	Italian	21	5.8	1 yr.
L. D.	5 yr.	Italian	14	0	5 mo.
E. C.	11 yr.	American	24	3.1	18 mo.
D. E.	11 yr.	American	8	4.3	1 yr.
P. H.	10 yr.	Irish	9	1.5	6 mo.
G. O.	16 yr.	American	4	28.1	3 yr.
		II. Base-Forming Ketogenic Diets			
G. R.	12 yr.	Italian	0	0	6 mo.
D. M.	10 yr.	Porto Rican	0	0.06	1 yr.
J. R.	7 yr.	American	0	0	3 mo.
W. M.	4 yr.	Portuguese	0	0	5 mo.
F. J.	12 yr.	American	5	0	2 yr.
L. S.	12 yr.	Russian	5	6.8	2 yr.
T. P.	11 yr.	Italian	2	2.0	2 yr.
F. J.	8 yr.	Irish	1	5.0	8 mo.
S. L.	9 yr.	American	0	1.0	6 mo.
R. C.	6 yr.	American	0	1.2	6 mo.

Comparative studies of the daily dietaries and the individual idiosyncrasies of children revealed nutritional factors related to the instance of dental caries.

Quantitative estimates of the daily food intake of the children hospitalized for weeks and months for chronic disturbances and subsequent follow-ups at home by a trained dietitian offered comparative data regarding the dietary content of each of the children studied from the standpoint of dental caries. The current standards of daily requirement in each of the nutrients were considered as base lines for the determination of proficiency or deficiency in a particular nutrient or vitamin. The results of these determinations are presented for each nutrient in terms of the groups immune and susceptible, respectively, to dental caries.

Both immune and susceptible groups showed no deficiency in any of these nutrients. Two of the children with extreme caries were allergic and were deprived from adequate intake of milk and other sources of animal protein. The group immune to caries consumed a daily carbohydrate no greater than 25 per cent of the total caloric intake, and none showed what might be considered strikingly low carbohydrate intake for children. Seven out of fifty-two with a moderate amount of caries consumed a relatively high amount of carbohydrate.

DEGREE OF DENTAL CARIES AND DAILY DIETARY

LIFE CARIES INDEX	PR.		F.		C.		ACID	BASIC	Ca		P		VIT. A		VIT. B		VIT. C		VIT. D	
	A	D	A	D	H	A			A	D	A	D	A	D	A	D	A	D	A	D
0	13	0	13	0	5	8	1	12	11	2	13	0	13	0	11	2	11	2	13	0
0-15	52	0	52	0	7	45	17	35	42	10	35	17	50	2	25	27	45	7	30	22
15-	33	2	35	0	6	46	26	9	22	13	26	9	34	1	18	17	27	8	13	22

Pr=protein.
F=lipids.
C=carbohydrate.
a=adequate.
d=deficient.
h=high.

On the other hand, six of these with extreme caries were maintained on abnormally high carbohydrate diets. Carbohydrate has been thus far heralded as a chief offender of caries because it ferments to lactic acid which subsequently dissolves the enamel, but also because it favors the formation of plaques under which caries takes place.

Sugar itself does not produce caries, for tooth cavities have been filled with acidophilus organisms and sugar and sealed tight without caries formation. Boyd and Drain were able to arrest dental caries in diabetic children on a low carbohydrate diet, but the sugar content of the children's diet was necessarily ruled out as the destructive factor in caries. They observed that children with celiac disease maintained on dietaries adequate in all respects but containing relatively large amounts of simple sugars responded equally as well as the diabetic children so far as the arrest of caries was concerned.

The Mellanbys continue to place particular stress on the carbohydrate content of the dietary in its relation to the formation of dental caries. We were led to conclude that the ill-effects of high carbohydrate feeding are chiefly the result of the diminution in the dietary of more important nutrients rather than in the harmful effect of the carbohydrate as the nutrient.

Children both immune and susceptible to caries, respectively, showed no dietary disturbance in vitamin A content. In a subsequent study of the relation of vitamin A to infections in children, we have neither observed that the deprivation of vitamin A for several weeks nor the administration of excessive amounts of standardized carotene produced any change in the dental caries of these children. These clinical observations are contrary to those Marshall made in experimental animals. Apparently there is a considerable gap in the applicability of experimental results in animals to human beings. Bloch's observations of Danish children with xerophthalmia revealed no dental caries related to this extreme vitamin A deprivation.

Children with moderate and extreme degrees of caries showed a diminished intake of vitamin B complex, but it is important not to attribute the deficiency to this factor as directly causative of dental caries. This is particularly emphasized by the fact that dental caries is not a concomitant of beriberi or pellagra in countries in which these deficiency diseases are endemic. The nature of the dietaries of the children with caries is such that vitamin B deficiency is a consistent observation, but rather indirect in its causation of caries.

Few children susceptible to caries showed any striking diminution in the daily vitamin C intake. We cannot attribute dental caries to lack of vitamin C; else the vast number of children with dental caries throughout the world are suffering from latent scurvy. The guinea pig, most sensitive to vitamin C deprivation, shows microscopic lesions in the teeth as the first sign of scurvy. But these animal observations are not applicable to human beings. Hess observed no unusual amount of caries in children suffering from scurvy in infancy. Jones reported widespread caries in children in Hawaii where tropical fruits, abundant in vitamin C, are the staple articles of food throughout the

year. The Eskimos consume minimum amounts of vitamin C foods, and yet their teeth are the best among the peoples of the world. Hankes' observations that a pint of orange juice a day is protective of dental caries is in the light of our present studies interpreted not on the basis of this vitamin C content, but rather on its considerable alkali-forming content. In a sense, the observation of the Mellanbys of the carries-producing effect of carbohydrate may be interpreted in the light of this work as the result of its considerable acid-forming content in the dietary of growing children.

The children of both immune and susceptible groups, respectively, showed no dietary deficiency in vitamin D. Although the group free from caries was simultaneously free from rickets in infancy, 20 per cent of the group with moderate and extreme caries had rachitic manifestations clinically or chemically or both, demonstrable in infancy. On the other hand, the greater number of children with caries showed no clinical evidence of rickets during infancy and were given adequate amounts of cod liver oil daily during infancy and yet developed a marked degree of caries both in the deciduous and in the permanent teeth.

We concluded with Hess that dental caries is not brought about solely by rickets, but rather it is one of several etiologic factors. The Mellanbys, however, have stressed the determining rôle of vitamin D in the production and in the prevention of dental caries in puppies as well as in children. In our group of children rickets was a contributory but not the determining factor in the development of dental caries.

It must not be forgotten that rickets affects not only bones but also the muscles, which by virtue of their hypotonicity remove the natural stimulus of development of the long bones as well as of the maxilla. Various observers have recorded brittleness of deciduous teeth, which has only been occasionally our own experience in severe rickets. Others have observed defective formation of the permanent teeth which in our own group we have been unable to relate, possibly because of the present rarity of severe rickets in our selected group of children with dental caries.

Children free from dental caries showed consistently a dietary excessive in alkali-forming minerals. Children with moderate caries showed a deficiency in alkali-forming salts and a preponderance in the acid-forming constituents of the diet. Paralleling the mineral character of the respective dietaries of these two groups of children was the preponderance of intake of raw fruits and vegetables in the caries-free group in comparison with occasional raw food intake in the high caries group. We have come to the conclusion on the basis of these observations that dental caries is a disease of mineral metabolism particularly disturbed during the peaks of rapid growth and development of childhood. These conclusions find strong confirmation in the work of Hess, Boyd and Drain.

Most children maintained on base-forming dietaries containing ample amounts of raw base-forming foods at each meal were resistant to the development of dental caries. These conclusions have been further reinforced by a parallel study of epileptic children under strict dietary supervision for months

at the hospital and for years at home. These young patients were treated with various types of ketogenic diets for the alleviation of their convulsive seizures.

Examination of the life caries index of each of these children resolved them into two groups, one with low life caries indices, and the other with very high indices. And these two extremes, representative of the relative degrees of dental decay, paralleled very strikingly the mineral nature of the ketogenic diets. It is to be remembered that all ketogenic diets produce chronic ketosis and dehydration in all bodily tissues and organs of these children. But some of the ketogenic diets may be base-forming and others acid-forming in mineral intake, and both nevertheless produce varying degrees of chronic ketosis.

This necessarily is productive of some degree of systemic decalcification of bones and teeth. Those children maintained on a ketogenic diet whose mineral intake was acid-forming showed marked dental caries, while those children maintained on a ketogenic diet whose mineral intake was base-forming showed low life caries indices. Thus we have confirmatory clinical and biochemical evidence of the rôle of mineral metabolism as it affects the course of teeth during growth and development.

Absolute immunity to dental caries is nonexistent, but certain races reveal so little dental decay in comparison with its enormous incidence among civilized peoples that practical immunity may be claimed with good reason. Studies of the geographical distribution of disease furnish mass means of testing hypotheses. The dietaries of primitive races are known, so it is logical to determine the common basis, if any, for the relative immunity to caries.

The diets of all natural races are ever so much more varied than those of civilized races. Although certain articles form the staple food in certain areas, yet in no case do they constitute the sole food of any race. Another feature besides variety in foods is sapidity. There is a constant inclusion of raw and steamed articles of food which have a direct stimulating effect upon the salivary glands. This is common to dwellers in the arctic and equatorial regions, in the meat-eating and in the vegetarian tribes, in the low types, and in the high types of uncivilized man.

Foods most frequently recurring among all races immune from caries are alkaline-forming fruits and berries, balancing the fresh fish and raw meat. Most of the dietaries are base-forming, while some are acid-forming but both types in all races are complete in every nutrient respect and ever abundant in mineral requirement.

A constant characteristic of all dietaries of races immune from caries is the minimal interference with food as obtained in its natural state. The greater portion of the food consumed, whatever it be, is necessarily raw and at the most steamed. Cooking of food in primitive races compared with the civilized parallels in a sense the minimal and extreme instance of caries.

Another striking property of primitive food is its masticatory value. Nearly all races relatively immune to caries show marked attrition of the teeth. This is not due to the coarse or gritty nature of the food, for native races are most careful in the preparation of their meals. Attrition is con-

sidered more probably due to the mechanical effect of fibrous particles combined with the constant acid action of fruits and berries. Primitive dietaries bearing these characteristics are so definitely preventive of dental caries that a sudden change in the dietary habits of races for those preferred by civilized peoples is rapidly productive of caries.

CONCLUSION

Dental caries is a syndrome of many systemic and local disturbances rather than a simple disease of single etiology.

Determination of the degree of dental caries was made by means of Bödecker's life caries index in children under continuous supervision for other systemic conditions.

Children free from dental caries were of the more stable elements of their race whose general and mouth hygiene were superior to children with marked dental caries.

Dental caries is maximum at six years for deciduous teeth and at twelve years for permanent teeth, the incidence increasing rapidly with age. This rhythm parallels that of development, thus relating dental caries to deep-seated metabolic changes in the growing body.

The degree and prevalence of dental caries are unaffected by recurrent mouth and upper respiratory infection.

Dental caries is neither prevented nor minimized by breast feeding. Children free from dental caries were maintained in infancy on base-forming dietaries containing early additions of semisolid food in the first months of life.

Children with moderate and extreme dental caries showed inadequate intakes of vitamins B, C and D.

Children maintained on ketogenic diets, base-forming in their mineral content, showed low life caries indices; while those maintained on ketogenic diets, acid-forming in their mineral content, showed marked dental caries.

Children free from dental caries showed consistently a dietary intake excessive in alkali-forming minerals with a preponderance of raw fruits and vegetables.

Races immune to dental caries consume sufficient raw alkali-forming fruits and berries to balance the acid-forming fresh fish and raw meat.

THE TEN POINT PLAN FOR THE CLINICAL CONTROL OF CARIES IN CHILDREN

1. Dental caries is a syndrome of systemic rather than of local disturbances, individual for each child.
2. Determine body build in relation to parents, and correct deviations in glandular function by endocrine therapy.
3. Immunize against contagious diseases, and eliminate chronic loci of infection to protect from metabolic deficiency.
4. Evaluate metabolic efficiency from food tolerance, digestive power, and alimentary waste to improve nutrient utilization.

5. Ascertain dietary habits to supplement acid milk, raw fruits and vegetables, and viscera, and thus shift to the alkaline-forming regime.
6. Order calcium phosphates and viosterol administered with raw fruit juices between meals.
7. Supervise the mineral and vitamin adequacy particularly during the periods of rapid growth—infancy, second dentition, and pubescence.
8. Encourage chewing coarse food to stimulate the muscles of mastication and the gums.
9. Enforce mouth hygiene routinely, including proper brushing of the teeth after each meal.
10. Advise periodic dental and physical examination and treatment, semiannually after the second year.

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ORAL CANCER

PATHOLOGY, CLINICAL FEATURES, DIAGNOSIS

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CARCINOMA of the oral cavity presents the interest associated with a disease which is always of a serious and ofttimes of a fatal nature. The diversity of the tumor types will always make the pathology of the region an alluring subject. The problem of treatment is complex and difficult. Here it is that the comparison between the results of irradiation therapy and surgical excision are brought down to a rather fine point. The older methods of surgical attack have been forced to go through a transition because of the remarkable development of newer irradiation technics. In a way, however, a greater responsibility has been placed upon the surgeon, first, because of the need for a more careful selection of cases for surgical care, and, second, because of the development of the so-called "surgery of access" necessary for the application of radium in certain locations and under certain conditions.

In the present paper only malignant tumors of the oral mucosa proper will be discussed. The part of the tongue posterior to the circumvallate papillae will be discussed in a subsequent paper along with malignancy of the pharyngeal region to which it more closely corresponds pathologically and so far as reaction to treatment is concerned.

Incidence.—Quick saw 473 cases of cancer of the tongue in 2,741 cases of intraoral cancer. In Scotland, Fraser found the rate of incidence for buccal cancer per million of population to be 154. In the United States in 1929 the rate of deaths from cancer of the buccal cavity per 100,000 population was 3.

Site.—Epidermoid carcinoma of the tongue is found most commonly along the lateral border of the tongue, but not uncommonly it appears first in or extends to the floor of the mouth. A carcinoma of the floor of the mouth is likely relatively soon to invade the tongue. Carcinoma of the dorsum of the tongue is not so common and, when it appears, is likely to be secondary to a leucoplakia or a luetic scar. Geschickter in a tabulation of the Hopkins series of tongue epitheliomas found 118 were located on the midborder, 60 on the undersurface, 56 at the base, and 38 on the tip.

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About 10 per cent of cancer of the buccal cavity proper appears about the salivary duct in the floor of the mouth. Fraser in 105 cases found that 43 per cent of buccal cancer appeared on the anterior two-thirds of the tongue, 20 per cent appeared in the palatoglossal sulcus, and 11 per cent appeared in the gingivoglossal sulcus. The remaining 16 per cent of Fraser's cases showed no constancy as to site. Of cancer of the tongue Fraser found that about 68 per cent appeared on the edge and 29 per cent on the dorsum. Primary carcinoma is very rare in the posterior tongue, but secondary invasion is rather common. Cancer of the tongue itself is more frequent than cancer of the floor of the mouth, gum, and cheek combined.

Sex.—At least 85 per cent of epidermoid carcinoma of the anterior two-thirds of the tongue and floor of the mouth appear in the male sex. In the Hopkins series 282 occurred in males and 27 in females.

Pathology.—Microscopically, practically all epitheliomas of the anterior two-thirds of the tongue are of the simple acanthomatous type. A very occasional

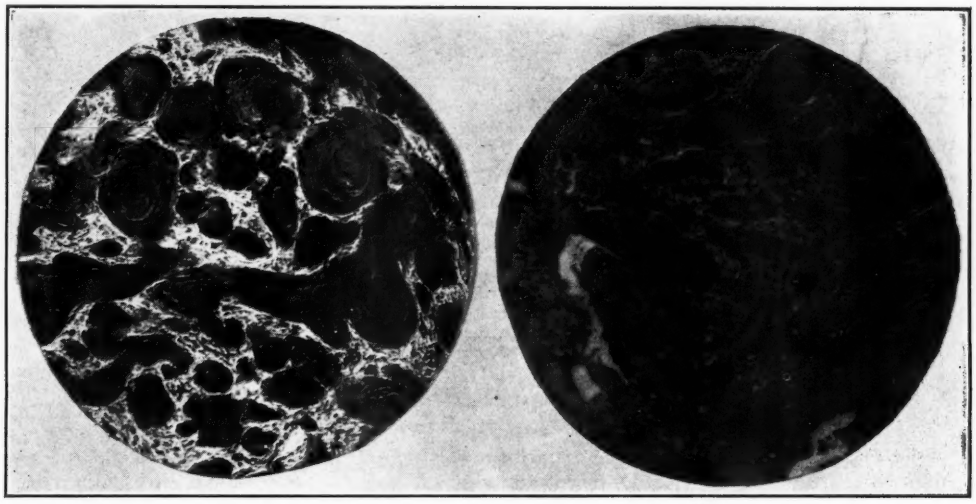

Fig. 1. Fig. 2.

Fig. 1.—Section shows irregular nests of fairly well-differentiated squamous epithelial cells imbedded in a very desmoplastic stroma—desmoplastic type of squamous cell epithelioma likely to be fairly radioresistant, Type I.

Fig. 2.—Section shows fairly adult squamous pegs infiltrating into a fairly dense granulomatous stroma. The epithelial cell nests are for the most part fairly well differentiated. Squamous cell epithelioma, Type II.

glandular carcinoma has here and there been reported. An alveolar arrangement without squamous cell characteristics, suggesting ducts of mucous glands, is rarely found. From the mucous glands or the buccal mucosa of the floor of the mouth the so-called transitional cell epithelioma may arise. This lesion is more fully described where it is more commonly seen—under pharyngeal epithelioma. The pathology is similar whenever seen. It may be difficult to determine whether a tumor arises from squamous or transitional cell epithelium. Because of metaplasia, squamous cells may appear in transitional cell tumors. On the other hand, squamous cell tumors may be anaplastic and fail to produce keratin. The pursuit of histogenesis is therefore often rendered uncertain. Most often acanthoma of the tongue presents fairly adult flat epithelium with hornified concentric flattened groups of cells—the so-called "epithelial pearls"

and intercellular fibrils. (Figs. 1-4.) The less adult cells are seen in the form of cords of opaque polyhedral cells with hyperchromatic nuclei. In the more malignant type the cells grow more diffusely. On the whole, however, epithelioma

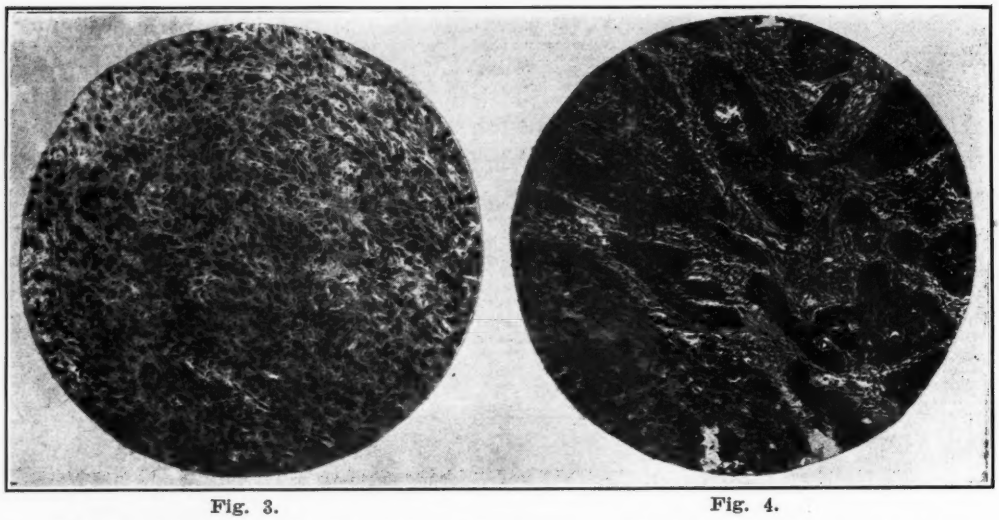

Fig. 3. Fig. 4.

Fig. 3.—Section shows a fairly diffuse squamous epithelioma which is moderately desmoplastic. The tumor cells are quite undifferentiated, vary considerably in form and size, and for the most part have a spindle cell arrangement. The tendency to desmoplasia may mean that the tumor tends to be somewhat resistant to irradiation. Broders' Type IV.

Fig. 4.—Section of a baso squamous epithelioma showing irregular branching nests of spindle form epithelial cells imbedded in a fairly dense fibroblastic stroma. Other areas show definite squamous cell arrangement. The majority of the picture is made up of irregular branching masses of basal cells.

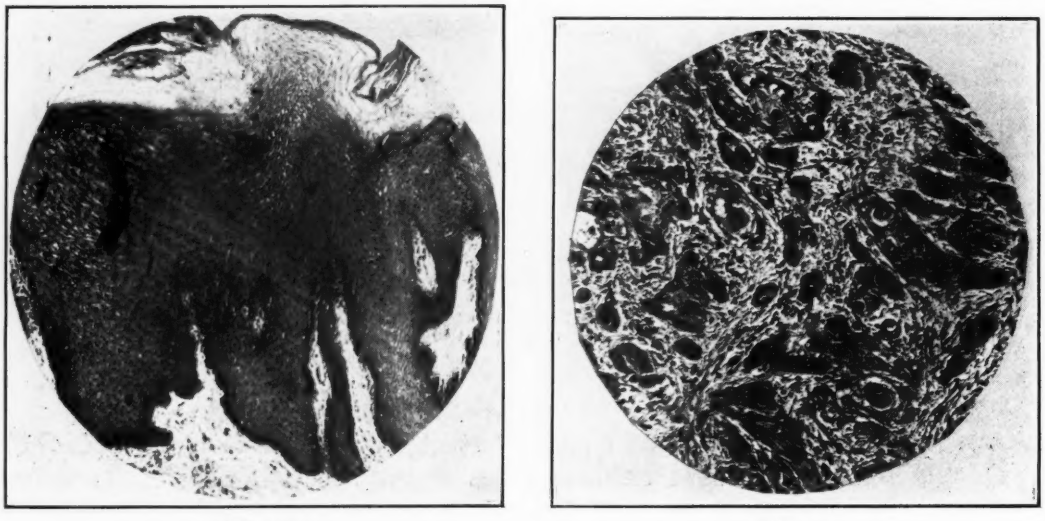

Fig. 5. Fig. 6.

Fig. 5.—Section of a benign squamous papilloma showing broad based epithelial pegs made up of adult squamous epiderm. The cutis is hyalinized and dense. No atypical penetration is seen.

Fig. 6.—Section from metastatic lymph node showing irregular squamous epitheliomatous nests filtrating throughout loose connective tissue stroma. A weak attempt at low grade pearl formation is occasionally seen. The cells are only moderately well differentiated.

of the anterior two-thirds of the tongue exhibits a fairly uniform microscopic picture. The down growth of epithelium in the early stages is almost always preceded by lymphocytic infiltration and edema of the submucosa. Soon a

considerable proliferation of fibroblastic tissue develops and surrounds the invading cells. As the proliferating cells travel along the lymph channels, the muscular tissue of the tongue is fairly rapidly invaded. Although metastasis usually shows about the same structure as the local lesions, anaplasia, as a rule, is more marked.

Grossly epidermoid carcinoma of the tongue in most instances first presents itself as an indurated ulcer with hard walls (Fig. 4). Very rarely a deeper submucous nodule may be the first evidence of the malignant growth but soon this type breaks down and produces a deep ragged exacerbation. Epithelioma starting in a leucoplakia may at first appear as a broad flat induration, and necrosis may be relatively late. Following leucoplakia multiple areas showing carcinomatous degeneration may be found. Simple papilloma (Fig. 5) with carcinomatous degeneration or diffuse papillomatosis from which carcinoma develops in one or several foci in the latter case at least are rarer forms of the disease. Ewing has seen a very extensive lingual cancer without invasion of the lymph nodes in which the lesion consisted of a plexiform infolding of the entire epithelial layer without invasion by cell groups. Although, as a rule, epidermoid carcinoma of the tongue metastasizes relatively early to the tributary lymph nodes (Fig. 6), Ewing urges that papillary plexiform, superficially diffuse and leucoplakic lesions show metastasis rather late.

CLINICAL FEATURES

Early Period.—A knowledge of the appearance of an early malignant change is extremely important. A good many years ago Butlin described five clinical pictures which he considered the most frequent and typical of the early forms of cancer of the tongue.

1. A little plaquelike, hard sore, smooth and polished but neither ulcerated nor excoriated.

2. The transformation or replacement of a simple ulcer by a cancerous ulcer, which differs from simple ulcer only by feeling very little stiffer and very little firmer.

3. The transformation of an entire plaque of leucoplakia to a plaque of cancer. The difference is marked by very slight increase of induration but without excoriation or ulceration.

4. The transformation of one small area of a leucoplakic tongue into cancer marked by only very slight and superficial hardening.

5. A white warty growth or compound wart neither broken nor ulcerated and feeling at first as if it is fixed to the mucous membrane and quite superficial.

"Of all the actual beginnings of cancer, there is none nearly so important as a wart or warty growth, especially when this forms on the surface of a tongue which is the seat of one of the chronic conditions which predisposes to the occurrence of cancer." (Butlin and Spencer.)

The earliest sign of malignant degeneration is increasing induration. Ulceration is the next earliest sign, and it is present in most instances so soon that it must be taken to be an almost necessary sign. The type of carcinoma that begins as a hard induration beneath the epidermis does not ulcerate immediately, but the form is a rare one. Papillary growths of the warty type may also ulcerate rather late.

In the early stages growth may be quite indolent, and several months may intervene with little progress shown. But the tendency to heal is not present, even after removing all evident sources of irritation, such as jagged teeth, rough bridges, or crowns. Finally, at a variable period of from two to four to five months the progressive character of the growth becomes evident. The increasing induration, the greater ulceration, and the increase in size of the whole of the lesion are all evidence of the character of the growth expressed in terms that are hardly to be mistaken. As the growth becomes larger, three or more forms are seen. One, a form showing increasing induration and ulceration without fungation; two, a type tending to fungation externally and less infiltration of the surrounding tissues; and three, a form developing in a fissure or forming a fissure with an ulcerated base and induration of the walls of the fissure. The induration is a hardness described as "stony" which is quite characteristic. In

Fig. 7.—Photograph of midline carcinomatous ulceration of the tongue. There are three factors of particular interest in this patient. First, it is a carcinoma in a woman thirty-seven years of age, which is a rather early age for the appearance of cancer. Second, the lesion is located on the dorsum of the tongue, which is not very common. Third, only about 15 per cent of carcinomas of the tongue appear in women.

certain instances the ulceration overshadows the induration to such an extent that the diagnosis may not be very definite clinically.

Although some discomfort is usually present, pain cannot be regarded as of much diagnostic significance. It is frequently entirely absent. Later, of course, as the deeper structures are involved, pain is a quite constant symptom.

Mid Period.—For convenience a second state may be considered to have taken place when the diagnosis is obvious, at least, to the experienced observer. (Fig. 7.) The characteristics described in the first period all continue in their development. The induration and the ulceration progress. Epitheliomas surrounded by a wealth of fibrous tissue may show considerable central contraction with the ulcer in depression and the edge rolled rather high and feeling stony hard to palpation. On the other hand, the progress of the ulceration may be the outstanding characteristic. The indurated wall breaks down rather rapidly,

and soon a marked cavity is formed with a red granular ulcerated base, often superficially covered with necrotic débris. Although the induration of the base and edges is absolutely characteristic, it is not so pronounced as when the ulceration is not so rapid. Characteristically in all epitheliomas of the tongue or oral cavity in general the edges are prominently rolled and are hard and not much undermined. The fungating ulcerating type is quite characteristic. The ulcerating surface projects above the surface and growth tends to be external as much as beneath the surface. Infiltration of the base and edges, however, is always definite even from the first and, as the tumor advances, becomes absolutely definite. This latter type of clinical growth is considered to represent a less malignant form of epidermoid carcinoma than the infiltrating form. The clinical course is somewhat less rapid. As previously stated under pathology, papillary plexiform superficially diffuse and leucoplakic lesions tend to run a more benign course than the deeply infiltrating and rapidly ulcerating types.

In the mid period, evidence of metastasis appears sooner or later. In a general way metastasis appears first in the underlying lymph nodes into which the area of the tongue involved drains most directly. It is impossible to forecast with certainty the involvement for the reason that when one group or channel is blocked with malignant cells, it is impossible to know the changed course of lymphatic drainage. When the local ulcer is on the anterior tip, on the root (posterior third) or extends to the median line, metastasis to the lymphatic nodes of both sides may occur or rarely even when the ulcer is definitely unilateral. As a rule, however, in the anterior body, or mid body and floor of the mouth, metastasis occurs only on one side. The median raphe of the anterior two-thirds of the tongue has very little lymphatic communication through it. The submaxillary lymph nodes or the upper deep cervical lymph nodes on the same side become enlarged and typically hard first, as a rule, in unilateral carcinoma of the anterior and mid portion of the tongue. The little glands between the geniohyoglossia muscles and the mylohyoid muscles are not often evidently involved. The deep-lying position of these glands probably has something to do with this, but it is also true that only a few carcinomas lie in the area which drain to them most readily.

Variations in the clinical picture in the midstage are due largely to the extent and direction of the growth. This applies to both the local lesion and the state of the tributary lymphatics. As muscular action is impeded, difficulty in swallowing and speaking increases proportionately. Of course, difficulty in eating tends to cause loss of weight. In this period a foul discharge appears and a putrid odor is added to the patient's breath.

Late Period.—In the final stage of buccal carcinoma, the symptoms and signs of the middle period increase. Metastasis to the tributary lymphatics becomes evident. A node becomes enlarged and hard, and then there is possibly a second node. Within a period of a few months the involved lymph nodes show evidence of perforation of the capsule by malignant tissue by becoming fixed to the surrounding structures. The size of the mass increases and finally the neck becomes more or less fixed by a hard, large, diffusely infiltrating mass. Eventually there is a tendency for central necrosis, and finally the skin is in-

vaded and perforated, after which a fungating ulcer appears. Finally, amid the stench, salivation, discharge, and pain, the patient is gradually exhausted by lack of sleep, difficulty in eating, sepsis, and the absorption of toxins of the disease. If the cancerous tissue involves a large artery, death may occur comfortably by the way of a large secondary hemorrhage. When inanition becomes complete and death occurs, the pathologist usually reports a terminal pneumonia as being present.

CARCINOMA OF THE JAW

The term "carcinoma of the jaw" is a misnomer as far as primary squamous cell epithelioma is concerned. Squamous cell epithelioma of the epithelium of the gums does soon secondarily invade the mandible or the maxilla, and the clinical picture may appear as if the tumor originated within the jaw (Fig. 8). As teeth are soon loosened, it is not uncommon to see a loosened tooth extracted by the unsuspecting operator and soon afterward a fungating tumor develop in

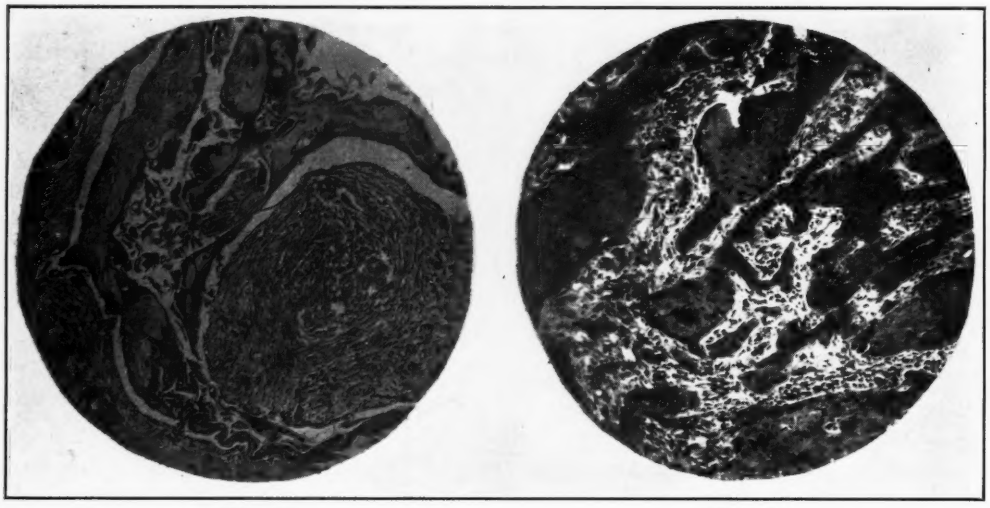

Fig. 8. Fig. 9.

Fig. 8.—Section of squamous cell epithelioma of the maxillary alveolar ridge showing penetrating masses of atypical epithelial cells. A considerable tendency to keratin formation is manifested, and the cells are fairly well differentiated. Type II.

Fig. 9.—Section of squamous cell carcinoma of mucous membrane of the cheek showing infiltration of the fibrous tissue stroma. Some tendency to keratinization and pearl formation is shown. Type II.

the socket which proves to be an epidermoid type of carcinoma. Microscopically, the malignant degeneration is there first, and the extraction of the tooth only activates the new growth. Probably the mandible is most often invaded by epithelioma originating in the lower lip. Epitheliomas of the cheek and floor of the mouth quite frequently sooner or later secondarily invade the bone. Metastatic nodes in the submaxillary region and a metastatic involvement of the buccal gland from an epithelioma of the cheek or the skin externally on the face also not uncommonly later result in secondary invasion of the mandible after the carcinomatous cells have perforated the capsule of the enlarged lymphatic nodes. In 2,741 cases of intraoral carcinoma, Quick found 149 to involve the inferior maxilla and 122 to involve the superior maxilla. It is doubtful, however, whether the frequency can be accurate; for very often secondary involvement is not mentioned when the primary involvement is from the neighboring structures.

EPIDERMOID CARCINOMA OF THE CHEEK

Epithelioma of the cheek is ordinarily of the squamous cell variety and fairly well differentiated. The microscopic picture is that of a somewhat more adult epithelioma than that described for anterior tongue and floor of the mouth, although the difference is not always pronounced (Fig. 9). Practically all the same irritative factors act on the tongue, the anterior palate, and the cheek. Leucoplakia not uncommonly involves the epithelium of the cheek as well as other epithelial tissue of the mouth. Papillary warts are also seen on the cheek (Fig. 10). Chewing tobacco and smoking probably play the same rôle as in cancer of the tongue. Transitional cell epithelioma (Figs. 11 and 12) very rarely may be seen in the cheek and more rarely a basal cell type of lesion.

Incidence.—As to frequency, in Quick's series of 2,741 cases of intraoral cancer, the mucosa of the cheek was involved 185 times. Fraser estimates 2.9 per cent of intraoral cancer as appearing in the cheek.

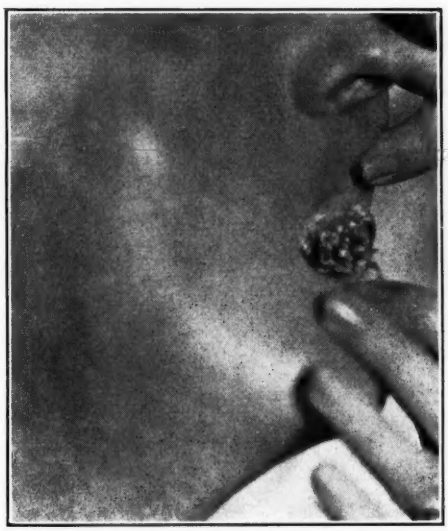

Fig. 10.—Photograph of patient with a large papillary wart of the cheek which showed tendency to recur after radical destruction. One would suspect an eventual tendency to malignant degeneration.

Clinical Features.—Clinically Ward and Smith describe three varieties, all of which may be seen: (1) a whitish papillary area with beginning induration; (2) an indurated mass in the substance of the cheek which eventually ulcerates; (3) an ulcer with hard edges infiltrating the substance of the cheek. The first type may grow rapidly, becoming papillary in form, and tending to bleed easily. There are, however, at least two other clinical types of lesions which are seen and are quite characteristic. (1) A whitish, warty, thick, elevated area with ulceration and induration in one part of it. (2) A fungating type of ulcer which projects above the mucosa of the cheek with a base which is not so indurated as the infiltrating type with a hard edge described by Ward and Smith. The ulcerative type is the commonest.

Metastases are only moderately early. The buccal gland in the cheek is likely to be involved. In the anterior cheek the submaxillary gland is most likely to be the site of metastasis. In the posterior cheek the upper deep cervical

nodes may be involved early. Infiltrating carcinoma of the posterior cheek and gingival sulcus soon infiltrates the muscles of mastication, limiting the

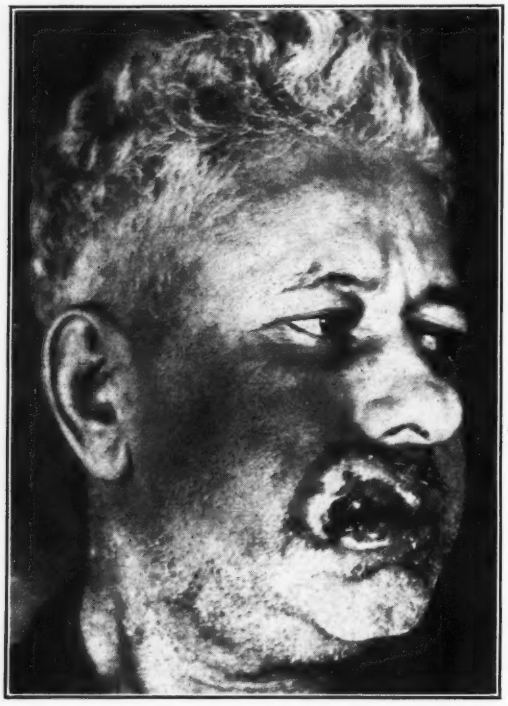

Fig. 11.—Photograph of a patient with a very rapidly growing carcinoma of the anterior cheek and angle of the mouth. This patient was rather young for a cancer of the mouth to develop—thirty-seven years of age; and as is characteristic in the more youthful, the local lesion was very malignant, grew rapidly, and soon metastasized to the neck glands. He was given intensive irradiation treatment but died about three months later.

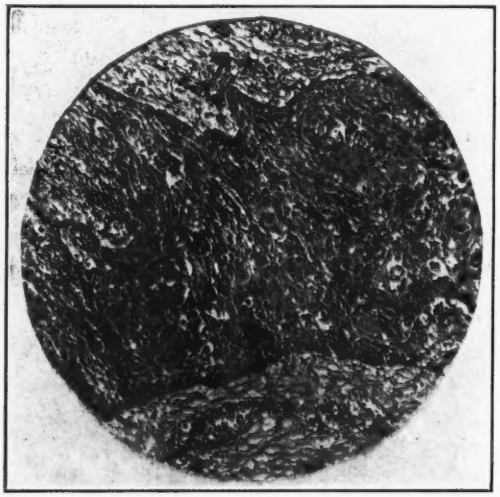

Fig. 12.—Section from the case in Fig. 11, shows rather highly undifferentiated columnar nests of anaplastic squamous epithelial cells. Occasional cells are fairly well differentiated, but for the most part they are quite anaplastic.

ability to open the mouth. After the muscles of mastication have become involved, the lesion takes on a more rapid growth. Clinically, as the location of

the growth progresses posteriorly, the malignancy seems to increase. Anatomic factors probably have some influence in causing this phenomenon. The cheek, being of no great thickness, is likely soon to be infiltrated and the external skin involved with an ultimate perforation. This may occur fairly early but usually it is a late occurrence.

EPIDERMOID CARCINOMA OF THE ANTERIOR PALATE

Epidermoid carcinoma of the anterior palate is usually a fairly adult type of squamous cell carcinoma. Some unusual features are present because of location, but on the whole it is somewhat similar to carcinoma of the anterior cheek. Metastasis to the regional lymph nodes is late. The lesion is fairly radioresistant. The pathologic picture and the clinical course of carcinoma of the soft palate are considerably different from those of carcinoma of the hard palate, and are related clinically more nearly to the more anaplastic growths in the posterior tongue, tonsil, and pharynx. The local ulcer on the hard palate assumes clinically (1) a papillary hypertrophic form, (2) a wide serpiginous ulceration with hard ulcerated edges, or rarely (3) a smooth globular swelling in which ulceration takes place in the center. The anterior palate metastasizes to the nodes into which the posterior palate drains. (Malignancy of the posterior palate will be discussed under pharyngeal tumors.)

DIAGNOSIS OF INTRAORAL CANCER

The diagnosis in the mid period or late period is practically always evident to the experienced observer. In many instances microscopic study as well as a study of the clinical picture aids the therapist in the proper selection of treatment. Preliminary biopsy, therefore, is recommended as a wise procedure in many situations. All questionable precancerous growths or early lesions in which the resulting deformity will be somewhat negligible are best handled by total excision and immediate microscopic examination. A very great misfortune may be suffered by the patient when a misinterpretation of what to do in a patient with a positive Wassermann reaction or other clinical signs of syphilis is made. When the diagnosis of the local lesion is questionable clinically, a positive Wassermann reaction or other signs of syphilis certainly do not warrant one in procrastination in trying the therapeutic test. If the patient has a carcinomatous lesion, much valuable time is lost, and it may well be that the only chance of the patient's life being saved is lost while an inexperienced therapist stands by and looks on. Certainly when in doubt of the nature of a suspicious lesion, an immediate biopsy is an imperative procedure. If negative for malignancy, the patient has not been harmed, and consideration may then be given to treatment of a lesion in which the factor of time is not so important.

As soon as the stage of ulceration has occurred, there are at least four types of ulceration which one should consider in making a clinical diagnosis of cancer of the intraoral region, namely, simple chronic ulcers, syphilitic ulcers, tuberculous ulcers, and cancerous ulcers. Actinomycosis rarely and even other fungus infections may also enter into consideration for differentiation. Certain borderline lesions of the chronic inflammatory type tax all the diagnostic resources of the most experienced men, and men of little experience are likely to find themselves uncertain when faced with relatively typical lesions.

Taken purely on the basis of percentage, it can be said with truth that the great majority of ulcerative lesions found in the mouth of patients over thirty-five years of age which have been present several weeks and which have shown little tendency to heal are of a malignant nature.

One may state without equivocation that in those of cancer age simple chronic inflammatory ulcers with some induration that fail to heal within two weeks following the removal of any evident sources of irritation, such as rough dental appliances or carious teeth, should be excised in toto and subjected to microscopic study. When such lesions heal rather promptly, however, one is justified in considering the lesion benign.

A primary lesion of syphilis in those of the cancer age is rare. One may suspect it when a nodule appears rapidly and is followed by a painless superficial ulcer whose base is a dull red copper color with a glazed surface which exudes a watery discharge. Later the cardboard-like induration of the base and edges becomes evident, and the tributary lymph nodes become characteristically elastic without any tenderness. It is not unusual for the surface of the ulcer to be covered with a grayish membrane when no friction is brought to bear directly on the base. When the lesion is the primary one of syphilis, a dark-field examination may reveal the *Spirocheta pallida*. None but the most experienced observers, however, can be certain, for several other spirochetes also appear in the mouth. Of course, somewhat later the secondary signs of syphilis make their appearance. The diagnosis, however, should not await the appearance of secondary lesions.

In tertiary syphilis ulceration most commonly follows the rupture of a gumma, therefore, the punched-out ulcer. In rarer instances ulceration follows a syphilitic glossitis. A gumma of the tongue most commonly appears on the dorsum of the tongue, while an epithelioma is most commonly on the lateral borders. In a luetic ulcer the Wassermann reaction is positive in 90 per cent—a fact which in no way rules out carcinoma. Typically, a gummatous ulcer has a so-called "washed leather" base. Early its tendency is to be punched out. Later it may be stellate fissured. The induration of the base and edges of a gummatous ulcer is not nearly so hard or so pronounced as an epithelioma. Intraoral syphilis is usually accompanied by other signs of syphilis elsewhere, but such signs do not rule out cancer. An unbroken gumma or a deep epithelioma presents an ill-defined mass. Early it might very likely be impossible to distinguish by clinical means between the two. The diagnosis can be proved by excision and microscopic examination.

A tuberculous ulcer of the mouth is practically always secondary to tuberculosis of the lung. A primary tuberculous ulcer of the mouth is a surgical curiosity but apparently has occurred, as proved in the literature by three or four postmortem examinations. A tuberculous ulcer has little or no induration. The granulations are watery and pale, not granular and red as in carcinoma. The edges are sinuous and undermined instead of rolled and indurated. Pain is a more characteristic symptom of a tuberculous ulcer than in either the carcinomatous or the luetic ulcer. About the main ulcer miliary tubercles may be seen, especially when one presses down on the area with a cover glass. The tip of the tongue is a fairly common location for a tuberculous ulcer. A tu-

berculous nodule rarely may be noted before the ulceration starts. When one has eliminated syphilis, carcinoma, and the mycosis, and taken into consideration the clinical characteristics of the ulcer, tuberculosis elsewhere, one can usually be reasonably certain of the diagnosis but final proof may await the biopsy.

Lymphosarcoma, fibrosarcoma, and mixed tumors usually do not ulcerate until late. In the case of the first two the mass is not hard, and in the case of the latter the mass is rather definitely circumscribed and possibly lobulated and movable save at the point of attachment. Lymphosarcoma is soft and fleshy in appearance and not definitely encapsulated; while a fibrosarcoma is somewhat firmer but presents a distinctly infiltrating mass.

TREATMENT

In a subsequent article after the principles of irradiation therapy are discussed, the subject of the practical care of malignancy in this region will be covered in its various phases.

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A CASE OF GENERALIZED OSTEOMYELITIS WITH INTERESTING JAW INVOLVEMENT LEADING TO FORMATION OF PARODONTAL CYST

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THE case to be described throws further light on the pathogenesis of follicular cysts, supporting Lartschneider's[1] contention that perifollicular inflammation is an important factor in cyst formation. The patient had been admitted to the hospital numerous times over a period of about five years on account of multiple osteomyelitis from streptococcic septicemia. The mandible (ramus) was one of the bones involved, and x-ray pictures taken at different intervals were available showing how a follicular cyst of the parodontal type gradually developed in the part of the mandible affected by osteomyelitis.

CASE REPORT

L. K. boy, aged ten years.

Chief Complaint.—Pain and swelling over left ramus, with inability to open mouth.

Past History.—Patient with an irrelevant family history had a past history of bronchopneumonia, complicated by double otitis media when nine weeks old; later measles, mumps, and scarlet fever when five years old.

Present Illness.—He was admitted to the hospital four years ago, when he complained of pain in the right lower leg. On the day before entry he vomited, had a chill, and was almost delirious. He was treated by incision for subperiosteal abscess and acute osteomyelitis. After the patient had been irrational for several days, the swelling in the leg gradually subsided. He received a blood transfusion (250 c.c.) following which there was a sudden rise in temperature, concomitant with swelling and tenderness and a brawny induration on his left arm. The temperature assumed a septic type. Swelling in the left arm gradually became fluctuant, and drainage was instituted under gas anesthesia. A culture from the arm showed hemolytic streptococcus. Six days later the patient suddenly developed a large fluctuant mass over the right upper back, which was incised and drained. The temperature and pulse gradually subsided, though for some time he ran a septic temperature, ranging between 99.6° and 102° F. All incisions healed; blood cultures which had showed hemolytic streptococcus on two occasions became negative; the temperature subsided and remained essentially normal for about two weeks, when the patient was allowed to go home.

He was readmitted, however, five weeks later complaining again of pain in the right lower leg and swelling of the left arm. Clinical pathology was not remarkable except for the white blood count which was 20,400 on admission. X-ray pictures revealed marked destructive changes at the lower end of the tibia with

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periosteal thickening, which had the characteristic appearance of chronic osteomyelitis, involving also the lower third of the fibula. An x-ray examination of the left shoulder and humerus revealed chronic osteomyelitic changes of the upper half of the humeral shaft (Fig. 1). He ran a temperature, picket-fence in character, of 98° to 101° F. The pulse fluctuated from 108 to 120 and 130. The swelling over the lower portion of the right tibia became localized into an abscess which was incised, draining for a week or so a small amount of pus. A new swelling formed on the left leg, and the forearm, when x-rayed, showed an ovoid osteolytic area at the distal portion of the right ulna, with a marked periosteal elevation (Fig. 2).

After having spent some time at a convalescent home, he was readmitted complaining of a soft swelling over the left jaw (ramus). The left side of the face was about three times normal size, the swelling was painless and non-

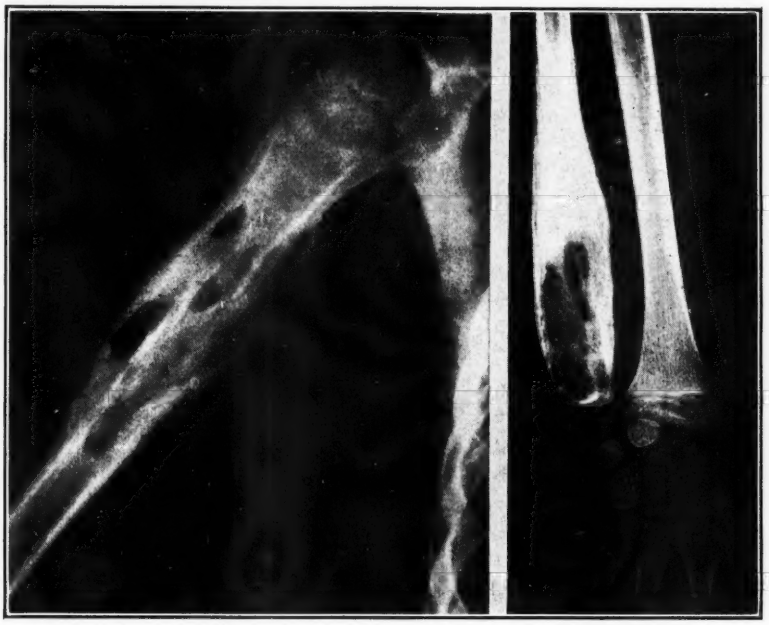

Fig. 1. Fig. 2.

Fig. 1.—Generalized osteomyelitis. X-ray picture of right humerus.
Fig. 2.—Generalized osteomyelitis. X-ray picture of left forearm showing involvement of the lower third of the ulna.

tender with a small discharging sinus on the upper surface, and there were palpable bilateral cervical glands. X-ray examination of the left mandible revealed some destruction of bone posterior to the second molar, consistent in appearance with osteomyelitis affecting the ramus (Fig. 3). It was believed that very little could be offered in the way of treatment at this time; so he was discharged home, condition unchanged.

A month later the patient was again admitted complaining of increased swelling over the left jaw and some pain over the right humerus. He ran a slightly elevated temperature. Urine was negative; hemoglobin 75 per cent; white cell count 12,800.

Roentgen examination of the mandible at this time (Fig. 4) showed marked osteomyelitis involving the entire ramus including the coronoid and condyloid

processes. The effect of the infection on the developing second molar was of particular interest. At the distal surface of the crown of that tooth was seen an area indicating enlargement of the bony crypt. This proved to be the beginning of a parodontal cyst formed from the epithelium of the enamel organ.

Present Condition.—Three years and four months after the onset of streptococcic septicemia with generalized osteomyelitis, and three years after involvement of the mandible, the patient returned to the hospital complaining of an increased swelling and pain in the left jaw. He was unable to eat because the jaw had gradually become "locked." An x-ray picture revealed a large cystic area involving the posterior part of the left side of the mandible. He was referred to the tumor clinic.

Examination.—Patient presented a slight swelling of the face on the region of the left ramus with evidence of a scar from a former fistula. He was unable to open his mouth more than one centimeter in the incisor region. The left condyle seemed ankylosed, and did not move in either lateral or forward motion. The teeth showed evidence of neglect, with several large cavities, especially in the left maxillary first molar which had an infected pulp.

His general condition was now greatly improved; all fistulas on involved parts had closed, and there was no swelling or tenderness of the bones. This seemed an opportune time for an operation on the jaw. Patient was referred to the x-ray department for pictures of the mandible and mandibular joint, and also to the medical department for serologic examination.

Laboratory Reports.—Hemoglobin 65 per cent; R.B.C. 4,500,000; W.B.C. 15,000; Kahn, negative; Hinton, negative; bleeding time, two and one-half min.; clotting time, two min.

X-ray Examination.—X-ray examination of the left mandible showed then (three years after its involvement) a large cystic area involving the posterior portion of the jaw, with the third molar displaced upward into the coronoid process of the ramus; changes indicating osteomyelitis were still present in the superior part of the ramus, particularly in the enlarged condyle (Fig. 5).

Diagnosis.—Parodontal cyst, follicular type, of the mandible; with osteomyelitis of the ramus and enlargement of the condyle causing partial ankylosis. Enucleation of the cyst was advised as the first step of treatment, to be followed later, when the mandible had become sufficiently strong and the osteomyelitis cured, by arthroplasty if still indicated.

Operation.—On account of the ankylosis, I extracted the maxillary first molar (pulpitis) and the mandibular first molar to obtain access to the posterior part of the mandible. I made the incision on the crest of the alveolar process extending along the anterior part of the ramus. The cyst, which according to the patient had ruptured and discharged into the mouth two days before the operation, was then enucleated. The displaced third molar was removed, and the second molar from which the cyst had originally formed was allowed to remain after all parts of the cyst membrane attached to it were removed. I have found this procedure[2] successful in other cases of young patients. The elimination of the pressure from the cystic contents generally allows the tooth to erupt and grow into its normal position. Recovery was uneventful: no in-

fection of the bone set in as was feared, the tooth continued to develop, and the cavity filled in slowly with bone. Ability to open the mouth was somewhat improved as a result of manipulation during the operation, but will have to be treated by arthroplasty later.

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 3.—Generalized osteomyelitis. X-xay picture of the mandible showing slight involvement of ramus posterior to developing second molar.

Fig. 4.—Generalized osteomyelitis. X-ray picture of mandible five months later showing effect of osteomyelitis of ramus on the developing second molar, the crypt of which shows enlargement on distal side. This proved to be the beginning of a parodontal cyst.

Fig. 5.—Generalized osteomyelitis. X-ray picture of mandible three years later showing osteomyelitis of ramus (condyloid process), and large cyst (parodontal type). Note third molar in coronoid process.

Pathologic Examination.—The specimen consisted of a fully formed tooth (mandibular molar) and one partly formed tooth (third molar), a piece of spongy bone, and pieces of soft, shiny, pink gray tissue which were smooth on

one surface and ragged on the other. These pieces seemed to be the lining of a follicular cyst. Microscopic examination revealed collagenic connective tissue, lined by a fragmented but not abnormal transitional epithelium. Both layers were focally and diffusely, though slightly, infiltrated with round cells. No malignant changes were noted anywhere.

Diagnosis.—Follicular cyst with chronic inflammation.

CONCLUSIONS

This patient presented a chronic osteomyelitis of the ramus, which no doubt stimulated cyst formation in the enamel organ of the developing second mandibular molar. The first x-ray film available (Fig. 3) shows the tooth developing normally in its crypt. The second x-ray film taken five months later (Fig. 4) shows a lateral expansion on the distal side of the bony crypt, the first indication of cyst formation. This location at the side of the tooth classifies it as a parodontal cyst. Three years after onset the x-ray (Fig. 5) shows that the cyst had developed to a considerable size, displacing the third molar into the coronoid process of the ramus.

This case is presented (1) to show a series of x-ray pictures illustrating the development of a parodontal cyst of follicular type, (2) to prove that follicular cysts may be caused by the stimulating effect of a local chronic infection such as osteomyelitis of the jaw.

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47 BAY STATE ROAD

CYST OF THE PAPILLA PALATINA

KURT H. THOMA, D.M.D., AND CHARLES A. BRACKETT,* BOSTON, MASS.

MUCOUS cysts occur in various parts of the oral mucosa; in the floor of the mouth—ranula, in the lip—labial cyst, in the tongue—cyst of the anterior gland, and in the palate. The latter frequently involve the papilla palatina forming in connection with a persisting nasopalatine duct, the palatal orifice of which becomes occluded through traumatic or infectious injury.

The region in which the papilla palatina cyst occurs is also the site of the incisive canal cyst and must not be confused with the latter. The first forms in the mucosa over the incisive foramen, the other in the incisive canal itself, involving and generally expanding the bone. It has been described by Kronfeld (1928), Schroff (1930), Thoma (1934), and Cohen (1936). It gives a characteristic osteolytic defect with cystic margin located in the median line behind the maxillary incisors (Fig. 1).

Gross Appearance, Physical Signs.—The cyst of the papilla palatina causes a round swelling gradually enlarging to the size of half a cherry. It forms in the soft tissue overlying the incisive foramen, expanding the papilla palatina as illustrated in Fig. 2. It is soft and fluctuant, but on palpation does not give the parchment-like sensation characteristic of the incisive canal cyst; though the latter when infected may perforate the cyst wall, making differentiation by physical examination more difficult. If sufficient pressure is applied on the swelling, discharge will exude from the cyst through the orifice of one of the small outlets of the nasopalatine ducts, which can be seen on careful examination as little occluded slits located on each side of the papilla. The discharge generally forms as semiliquid, mucofibrinous drops as illustrated in Figs. 4 and 5. The mucosa covering the swelling is usually not altered, as is the case in labial cysts and the true ranula, but retains its thickness and its rugae, though there may be redness du to inflammation causd by irritation of the mandibular incisors during mastication, or infection. In such cases the discharge may be of a purulent nature.

Roentgen Examination.—The x-ray picture helps to differentiate between the cyst of the papilla palatina and that of the incisive canal kind. In the latter a typical cystic area is seen in the film, as illustrated in Fig. 3, while in the former the roentgen examination is entirely negative, as seen in Figs. 6 and 7. In some cases the incisive foramen may be slightly enlarged, and occasionally there may be a little resorption due to pressure erosion.

Treatment.—Evacuation of the discharge will not cure the cyst as long as the cyst sac remains. It will refill with mucous secretion as soon as the opening of the duct again becomes closed or with fluid accumulated by transudation through the epithelial lining of the cyst. It is therefore necessary to remove the

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epitheliated sac and mucous glands by surgical method, or destroy them by means of a sclerosing solution, such as recommended by Cutler and Zollinger (1933) in gliomatous cysts. The latter method has proved successful in the two reported cases. The formula is as follows:

Absolute alcohol	6 c.c.
Chloroform	3 c.c.
Glacial acetic acid	1 c.c.
Ferric chloride	1 gr.

Fig. 1.

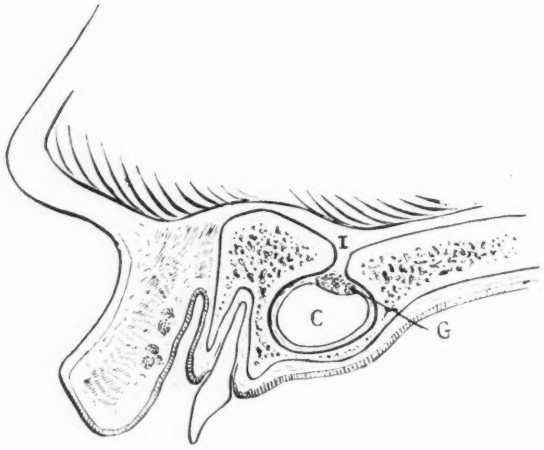

Fig. 2.

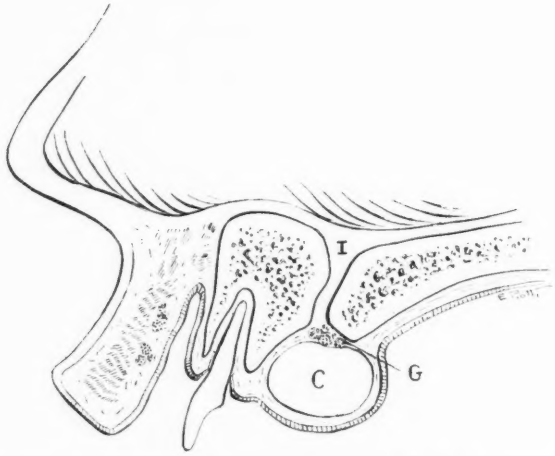

Fig. 1.—Incisive canal cyst, sagittal section through maxilla showing central location. *C*, cyst; *G*, mucous glands; *I*, incisive canal.

Fig. 2.—Cyst of the papilla palatina, sagittal section through maxilla showing extraosseous location. *C*, cyst; *G*, mucous glands; *I*, incisive canal.

CASE REPORTS

CASE 1.—H. C., a colored man, aged fifty-four years, married, complained of a swelling on the anterior part of the palate which prevented him from wearing his plate.

History.—Patient had an irrelevant medical history. All his teeth had been extracted several years before, and he had been wearing a rubber denture. Two years before, a painless swelling formed on the palate, discharged a salty fluid and disappeared. It came back several times, and now it was bigger than ever before. It caused pain in the maxilla and top of the head.

Local Examination.—The patient presented an edentulous maxilla with globular swelling on the anterior part, in the median line, involving the papilla palatina. It had the size of a cherry, was fluctuant, but the mucosa covering it appeared normal in every way (Fig. 4). When applying pressure a drop of gelatinous, mucoid discharge escaped from the orifice of the left nasopalatine duct, immediately adjoining the papilla. The orifice of the right duct was visible as an occluded small slit, but there was no discharge from it.

Fig. 3.—Incisive canal cyst, occlusal x-ray film.

Roentgen Examination.—The occlusal film of the maxilla showed no abnormal conditions except perhaps an abnormally large incisive foramen. There was no evidence of an incisive canal cyst (Fig. 6).

Diagnosis.—Retention cyst of the papilla palatina, with chronic infection.

Treatment.—With procain suprarenin anesthesia a semicircular incision was made along the anterior margin of the cyst. A jellylike, mucoid fluid escaped. This was cultured. A mucoperiosteal flap was detached from the bone which had attached to it the cyst sac. On account of the infection this had become very fragile and could not be detached in one piece. The bone of the palate was exposed, it presented a large incisive foramen 1 cm. in diameter. The flap, after being curetted and treated on the inner surface with the sclerosing solution, was reduced in size and replaced. A small piece of rubber dam was placed between the two sutures for drainage, and was removed the second day. The wound was irrigated daily with normal saline solution, and healed completely in seven days. The palate had become normal so that the denture could again be worn.

Laboratory Report.—The culture showed a moderate number of micrococcus catarrhalis, a moderate number of pneumococcus, and abundant streptococci.

CASE 2.—D. M., a young woman with irrelevant medical history, complained of a swelling on the hard palate.

Local History.—The patient had felt a soreness on the palate over a considerable period of time. The condition had become very much worse recently; the entire palate was painful to touch, making eating difficult, and there was

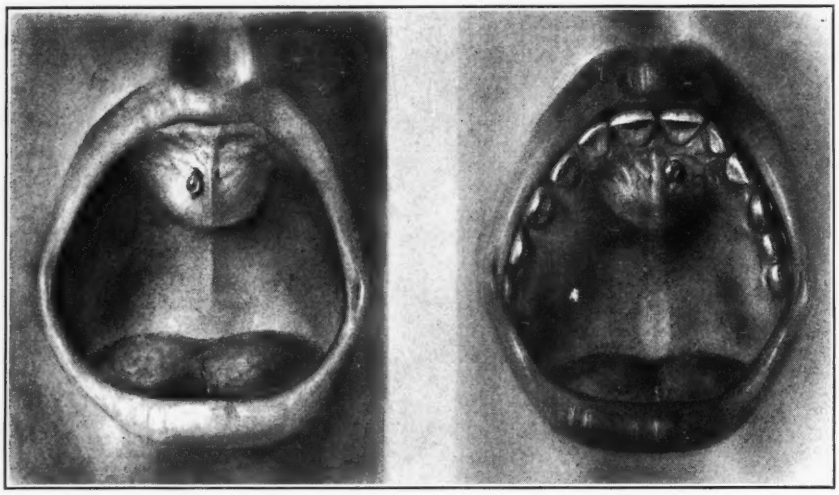

Fig. 4. Fig. 5.

Fig. 4.—Cyst of papilla palatina in edentulous maxilla. Mucopurulent discharge is seen at orifice of nasopalatine duct.

Fig. 5.—Cyst of papilla palatina with gingival inflammation. Pressure of swelling causing discharge through the orifice of the nasopalatine duct.

Fig. 6. Fig. 7.

Fig. 6.—Occlusal film showing edentulous maxilla without any abnormality of the bone.

Fig. 7.—Occlusal film showing normal teeth without any bony changes in anterior part of palate.

a salty discharge into the mouth. The gingivae bled easily due to trench mouth infection for which she had received treatment.

Local examination showed a semiglobular swelling on the palate centering around the papilla palatina, reaching from the incisors to the first molar region

(Fig. 5). The entire mucosa of the palate was red and inflamed; in both the maxilla and the mandible there was marked marginal gingivitis. When pressure was applied on the palatal swelling, a fibrinous, mucopurulent discharge escaped in drop form from the orifice of the left nasopalatine duct.

Laboratory Examination.—A smear taken from the gingiva showed on microscopic examination mixed pyogenic infection, without the presence of either fusiform bacilli or Vincent's spirillae. Examination of the discharge from the cyst showed mucoid substance with threads of fibrin and containing an abundance of pus cells. Occasional cocci were encountered.

Roentgen Examination.—An occlusal film taken of the maxilla showed no infected teeth and no osteolytic changes. Diagnosis of incisive canal cyst therefore could be excluded (Fig. 7).

Diagnosis.—Retention cyst of the papilla palatina, with infection.

Treatment.—With procain suprarenin anesthesia the contents of the cyst were removed by irrigation through the existing opening with a Luer syringe containing saline solution (blunt needle in syringe), and by applying pressure to facilitate evacuation of the fluid. After carefully isolating the part with gauze, the above described sclerosing solution was injected. (Care must be taken that the solution does not run over the surface of the mucosa, as it causes fixation and death of the superficial tissue cells.) The after-care consisted in daily irrigation of the cyst cavity, and the use of an astringent and antiseptic mouth wash. In ten days the mucosa of the palate was of normal appearance, and the swelling had entirely subsided.

CONCLUSIONS

1. Cysts of the papilla palatina form over the incisive foramen without involving the bone, and must be differentiated from incisive canal cysts.

2. For treatment of cysts of the papilla palatina the injection, after evacuation of all discharge, of a sclerosing solution gives satisfactory results.

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47 Bay State Road

INCISIVE CANAL CYST OF EDENTULOUS MAXILLA*

M. Michael Cohen, D.M.D., and Julius Levine, D.M.D., Boston, Mass.

INCISIVE canal cysts occur both in dentulous and in edentulous mouths; they are not of odontogenic origin. Schroff is of the opinion that they are derived from the nasopalatine duct or its remnants. The nasopalatine ducts are located approximately 2 cm. dorsal to the inner margin of the nostril and on each side of the nasal septum, where there is a slight depression. This depression leads into a small canal, the nasopalatine canal which is lined with epithelium, the lining of the inferior meatus. Each canal courses obliquely forward, and converges toward the nasal septum, descending almost vertically, and passing through the Y-shaped incisive foramen (anterior palatine foramen). The canals terminate in the roof of the mouth, where there may be found two very small slits on each side of the papilla palatina. These slits are often closed and impervious, but sometimes in adult life there is a communication between the nasal and the oral cavity via the nasopalatine duct. Rawengal made histologic studies of 27 cases. In 19 newborn infants, 5 had a continuous passage from the nasal to the oral cavities, 3 adults showed no passage, 14 had a blind sac from the nose, and 4 a blind sac from the mouth. Rawengal's studies also show that the epithelial cell rests, derived from the middle epithelial cord at the junction of the median primary plates and two lateral plates, may become enclosed in the incisive canal during the process of embryonic development. These cell rests may be stimulated to give rise to a cyst in later life. Noyes recently demonstrated the nasopalatine duct and canal by making serial sections of newborn infants. In no case was he able to show communication between the oral and the nasal cavities.

The immediate cause of incisive canal cysts may be trauma, from mastication or from ill-fitting dentures or removable bridges, causing irritation of the palatine papilla, and occlusion of the nasopalatine duct through inflammation. Activation of the mucous glands in the canal through any pathologic reaction may cause increased secretion and cyst formation in the incisive canal.

CASE REPORT

W. B., a fifty-two-year-old baker, with an essentially irrelevant medical history, complained of a pain and a burning sensation in the incisive canal region. All maxillary teeth had been extracted seven years before, and a vulcanite denture was inserted four weeks later. This was worn for one year. The patient during this time had a burning sensation in the region of the incisive canal. He was told to have a gold denture made because vulcanite was causing irritation of the mucosa of this region. This gave some relief, and was worn for four years. Because the patient still complained of pain, another dentist was consulted, who

*From the Dental Clinic of the Beth Israel Hospital.

advised him to have a denture of hecolite made, which he thought would give complete relief of the burning sensation. This new denture was worn for one year, then became loose, while the previous complaint of pain and burning persisted. He had a fourth denture made, this time again of rubber, with no relief of symptoms. During this entire period, no x-ray pictures were advised or taken, and the treatment consisted of local applications of iodine and the use of mouth washes.

Local Examination.—The patient, upon clinical examination, revealed a slight bulging of the buccal mucosa on the anterior part of the palate; the tissue seemed quite red and inflamed. Digital pressure revealed typical cystic fluctuation, and a tentative diagnosis of cyst was made.

Roentgen Examination.—X-ray pictures were advised and taken, and revealed a large circumscribed area symmetrically situated in the midline of the

Fig. 1.—X-ray picture of large incisive canal cyst.

palate in the region of the incisive foramen. From the x-ray examination, provisional diagnosis of incisive canal cyst was made.

Operation.—Under procaine anesthesia, an incision was made approximately the width of the cystic cavity, and the mucoperiosteum was elevated and laid back. This exposed bone of eggshell character, which covered the cyst wall. The eggshell bone was peeled off, and the denser bone was removed with the rongeur, exposing the cyst sac. The cyst was opened, and a thin, yellowish, brownish fluid was evacuated. The anterior wall of the cyst sac was then removed with a piece of tissue attached to it. The mucoperiosteal flap, instead of being sutured back to its original position, was turned in under the upper margin of the cyst cavity, and held there with an iodoform packing. This packing was changed every forty-eight hours for ten days, after which the edges had healed sufficiently, and the cavity was covered by mucosa, leaving a large depression open into the mouth. This was kept clean by irrigation with saline solution until it shrank to a shallow depression. The original cyst epithelium which

was left in the concavity, gradually took on the characteristics of the normal mouth epithelium. Part of the cyst sac was left in this case because of the close proximity to the floor of the nose, the danger of perforation of the nasal cavity, and in order to conserve the bone for good retention of the maxillary denture. Ten days after the operation, a plaster wash impression was taken in the patient's vulcanite denture, and the denture relined; giving the patient masticatory function, and facilitating keeping clean the cystic cavity.

Pathological Examination.—The removed tissue was fixed in formalin and sent to Dr. Kurt H. Thoma, Pathology Laboratory of Harvard Dental School, for microscopic examination. He reported on May 23, 1935, as follows:

The specimen consisted of several pieces of tissue, the largest of which measured 1.5 cm. × 0.5 cm. Microscopic examination showed a fibrous connective tissue sac made up of very fine collagen fiber bundles. Though it was not very vascular, it showed evidence of extravasation, which was probably due to operative trauma. This sac in places showed an epithelial covering which was of the squamous cell type, and contained in one place numerous mucous glands, surrounded by inflammatory cells which were absent in other parts of the tissue. The microscopic appearance of the tissue was consistent with that of incisive canal cysts.

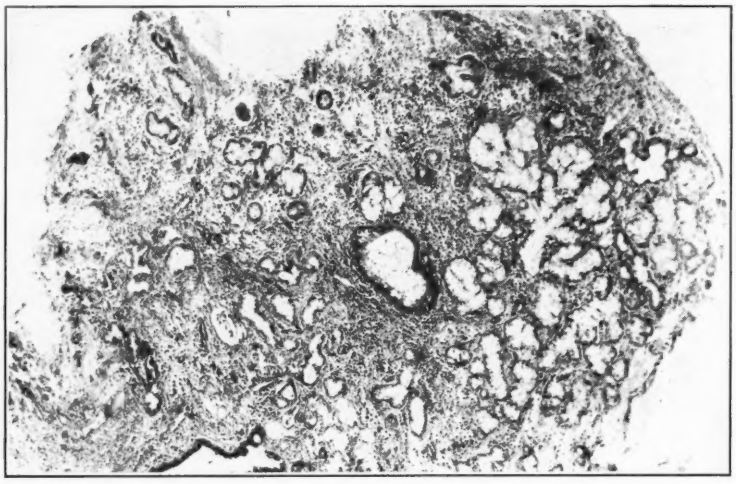

Fig. 2.—Photomicrograph of part of cyst wall showing mucous glands contained therein.

CONCLUSIONS

1. Repeated swellings of the palatine papilla should be looked on suspiciously, and a roentgen examination should be made.

2. X-ray pictures of the central incisor region, showing a large cystic area over the apices of the teeth, should be studied very carefully before treatment is instituted, because sometimes cysts of the incisive canal throw their shadow over the apices of the central incisors, giving the appearance of apical pathology.

3. In edentulous mouths with history of repeated swellings of the palatine papilla, or of pain radiating to the side of the nose or eye, radiographs should be taken to determine whether or not an incisive canal cyst is present.

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482 Beacon Street

THE USE OF SEDATIVES IN DENTISTRY AND ORAL SURGERY

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DURING the last few years there has occurred the development of innumerable drugs of a certain type, all of which tend more or less to produce the same desired effect. The intensity of this effect varies from a slight somniferous condition to that of a profound sedative action. To accomplish this premedication for the best interest of the patient requires a more or less complete knowledge of the pharmacologic and physiologic action of the present existing drugs that have flooded the market.

Before discussing the pharmacology and the specific physiologic action of these drugs, let us consider the patient. Is he to be an ambulatory case, that is, receive his treatments in a dental office, or is he to be hospitalized for an operative procedure? Whichever may be the choice, there will exist certain psychic factors which must be recognized. The mind and the body must be considered separate clinical entities and dealt with accordingly. Psychic trauma must be obviated completely in order to secure the advantage of working with a quiet, relaxed, and confident patient. To accomplish this, preliminary visits should be encouraged; hence the confidence of the patient is secured. Such visits should be used for a full discussion of the operative procedure contemplated and the anesthetic that is to be used, with its mode of application, so that any unpleasant or disagreeable previous experience may be erased from the memory. By means of such a conference, one understands better the make-up of the patient and his reaction to past unpleasant experiences, and can choose the preoperative drug more intelligently, bearing in mind any personal idiosyncrasies. This choice of sedation should be made in accordance with the temperament of the patient, special care being used for the apprehensive, imaginative, and hysterical person in order to guard against any additional psychic trauma that might result.

In choosing the preliminary medication the dentist should be well informed and have a complete knowledge of the chemical components and the pharmacologic and physiologic action of the drugs that he intends to use. He should not be forced by popular clamor to give and transfer allegiance, an allegiance stimulated by report of advertisement and not by proved worth. He must disregard fallacious, general impressions so easily grasped, and train himself to use the drugs the specific action of which has been determined through deliberate, unbiased, and untiring scientific study.

Attempts to employ barbiturates as general surgical anesthetics in human beings have not been uniformly successful. This is due, in part, to the fact that individuals vary in their reactions to them, and an individual may, himself, vary from time to time, depending upon his emotional state. Again, in human beings, barbiturates are really hypnotics and not true anesthetics. If sufficient quantities are given to insure complete anesthesia, in the majority, a period of

severe incoordination may ensue during recovery. It is now almost universally agreed that in surgery the barbiturates find their greatest field of usefulness, when employed in conjunction with other anesthetics, as preanesthetic sedatives, or, in other words, as a basal narcotic upon which may be supplemented local or general anesthesia. This basal hypnosis was first used in Germany twenty-five years ago with more or less success.

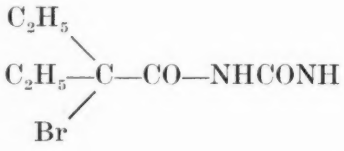
A warning should be inserted here that clinical science should recognize that so far as both manner and intensity are concerned, the action of a drug on man is not necessarily the same as the action on an animal; likewise, the action on the diseased is not necessarily the same as on the healthy man. Recently the *New England Journal of Medicine* (Vol. 210, No. 5, Page 275) issued a warning as to the use of barbiturates in obstetrical analgesia. So here, permit me to warn the members of the dental profession that the indiscriminate use by the patient of any of the following drugs should be checked before disaster occurs. By this I mean that these drugs *should be used only in operative cases where the patients will be under constant supervision.* With this proper supervision, the dentist may use any one of the following drugs as a preliminary medication to his operative procedure. His choice will more or less depend upon the accurate clinical data furnished him or, better still, on his actual past experience with the chosen drug.

Hypnotics are by definition, sleep-producing substances. Many groups of compounds, both organic and inorganic, have been found to possess the property of producing sleep to an appreciable degree, and have been suggested for use in medicine and dentistry. The barbituric acid series in addition to being relatively safe is capable of greater expansion from the chemical angle than the others, and it is in this field that most progress is now being made. It is here too that immediate interest has turned from hypnosis to the task of bridging the gap between simple sleep and surgical anesthesia. It is without question of primary importance to spare the surgical patient the physical agony of the operation; but if, in addition, we can avoid the mental agony of the periods before and after, we shall have taken still another step forward. It was with this purpose in view that the many pharmaceutical houses, several years ago, undertook the synthesis and study of a rather extensive group of branched chain barbiturates. This work was materially aided by the recent commercial availability of certain five and eight carbon atom alcohols derived respectively from natural gas pentanes by chlorination and hydrolysis, and from the pyrogenic decomposition of castor oil.

The pharmacologic studies on the recently prepared drugs involved years of study and detailed experimental work to arrive at a definite evaluation of these barbiturates. Most of these tests are made on small animals, namely, standardized albino rats and also on rabbits. Hence, it is comparatively simple to assess the probable values of a newly synthesized barbiturate. There are too many unknown factors, however, to permit an accurate guess as to the possible clinical effectiveness; this information can come only from experiments on the higher animals and from actual clinical tests on human beings. Few groups of compounds have been more intensively studied both chemically and pharmacologically than have the barbituric acids, yet in practice the different members

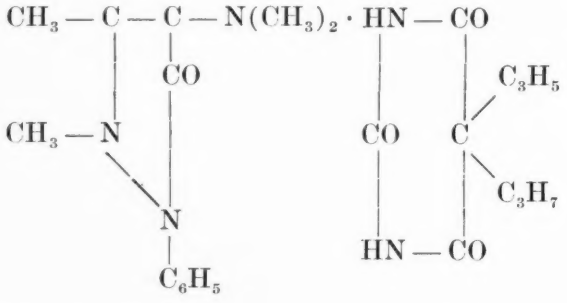
of the series are all too frequently employed indiscriminately. In order to acquaint ourselves with the drugs of the present day, we have tried out several of them in the dental, medical, obstetrical, and surgical clinics at the Boston City Hospital. This comprises a large series of cases from which to draw conclusions. These cases were all so-called house cases, that is, were patients in the hospital, and were returned to their respective wards and rooms after having received their operative treatments in the clinics; hence we were able to study the reactions of these drugs which have formulated definite opinions. No attempt will be made to show any partiality toward this or that drug, even though our actual clinical experience has shown that some drugs are more effective than others under certain varying conditions. The chemical structural formula of these drugs will be shown, but space will not permit going into the chemistry of them. They will be taken up alphabetically: (1) adalin, (2) allonal, (3) amytal (sodium), (4) avertin, (5) barbital (veronal), (6) dial, (7) empirin compound, (8) ipral, (9) luminal (phenobarbital), (10) nembutal (11) ortal, (12) sodium alurate.

Adalin.—Chemically known as brom-diethyl-acetyl carbamide with the structural formula:



This drug contains about 33 per cent bromine. It is a mild sedative and hypnotic and is particularly useful when powerful action is not desired. It is nonhabit forming. The average dose is from five to fifteen grains. It is an excellent sedative prior to dental operations which require a calm, cooperative patient devoid of restlessness.

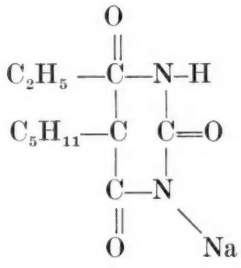
Allonal is a compound resulting from the fusion of allyl-iso-propyl-barbituric acid and amidopyrine (pyramidon) with the following structure:



It is about five times as effective as barbital (veronal), and twice as hypnotic as phenobarbital (luminal); yet in ratio to hypnotic efficiency it is relatively less toxic, and has a considerably wider margin of safety. Like all barbituric acid derivatives after absorption, it acts upon the central nervous system in the nature of a depressant. There is a rather prompt response with allonal. The resulting hypnosis is of four to five hours' duration which tapers off into a natural sleep. Apparently there is no cumulative action, thus ob-

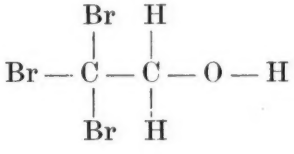
viating any so-called hangover, and there is a wide margin of therapeutic safety. Allonal has a definite place in oral surgery and dentistry; that is, to be used as a preanesthetic where novocaine is supplemented. In the clinic at the Boston City Hospital we generally give one to two tablets (one tablet contains one grain of allyl-iso-propyl-barbituric acid and one and two-thirds grains of pyramidon), which will diminish the apprehension and also a certain amount of shock.

Amytal (sodium) is the sodium salt of iso-amyl-ethyl-barbituric acid with the structural formula:



It is a hypnotic and sedative which is used orally in the preanesthetic preparation of surgical and dental patients. Experimental work on the toxicity showed the therapeutic index to be approximately two and one-half (2.5). It has some protecting effect against acute cocaine poisoning. Although sodium amytal has the same empirical formula as nembutal, the difference in the position of the methyl group is apparently responsible for certain differences in the physiologic effects. It requires about twice the dose of amytal to produce the same effect as with pento-barbital sodium (nembutal), whereas the recovery period is about double with sodium amytal as compared with nembutal. Dependence solely upon this drug for full anesthesia is not recommended.

Avertin is chemically known as tribromethanol and has the following structural formula:

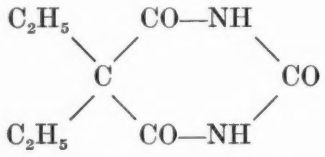


It is supplied commercially as avertin fluid, which is a solution of avertin in amylene hydrate. This solution is soluble in water and must be prepared at moderate temperatures (35° to 40° C.). One c.c. of the avertin fluid contains one gram of avertin and 0.5 gram of the solvent. It produces basal anesthesia and should be used as such, and has the advantage of having a protective influence against the toxic effects of local anesthetic drugs similar to that of the barbiturates. The relaxation under avertin alone is generally sufficient for minor operations, and is very serviceable in operations on the mouth, face, and neck because the anesthetist is out of the operative field. When electrosurgery is employed within the oral cavity, avertin is the anesthetic of choice. After

the administration of the avertin enema, the patient falls into a calm sleep and is spared any knowledge of the trip to the operating room. This absence of psychic shock is of special importance in impressionable subjects and children. The action of the drug is fairly rapid following installation into the rectum. It is absorbed by the intestinal mucosa more rapidly than the water in which it is dissolved. There is no local irritation of the rectal mucosa. Detoxication occurs in the liver through the formation of a compound with glycuronic acid, the resulting product being eliminated by the kidneys.

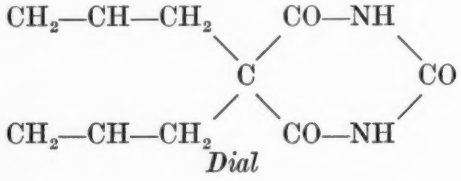
After absorption, avertin acts upon the central nervous system as do the commoner anesthetics. There are no deleterious effects upon the heart. A slight initial drop in the blood pressure occurs which is due to peripheral action, but this drop soon returns to normal. The liver and kidneys are not affected when normal conservative doses are administered. The respiratory center is influenced early, but the margin of safety is relatively great. The respiratory rate may decrease, but it is usually increased and often becomes shallow. In regard to the eyes, first the conjunctival and later the corneal reflex is abolished with the eyeballs fixed. The pupils react to light. There is an absence of nausea and excitement. The induction is unattended by salivation. Summing up the clinical appearance, we are led to believe that this type of anesthesia can be induced in an ideal manner. Once experienced, a patient will demand it again.

Barbital, the first barbituric acid derivative introduced to the profession in 1903 as a hypnotic and sedative, was called veronal, which is di-ethyl barbituric acid with the following structure:

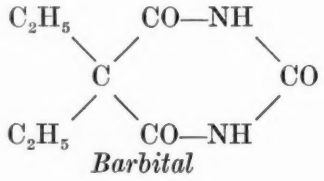


It is less potent than some of the other drugs and, if it is properly used, is one of the safest of hypnotics. Also if this drug is given postoperatively, it will relieve excitement and diminish pain. The average dosage is five to ten grains.

Dial is di-allyl-malonyl-urea or di-allyl-barbituric acid, which is closely related to barbital as seen by the formula:



Dial



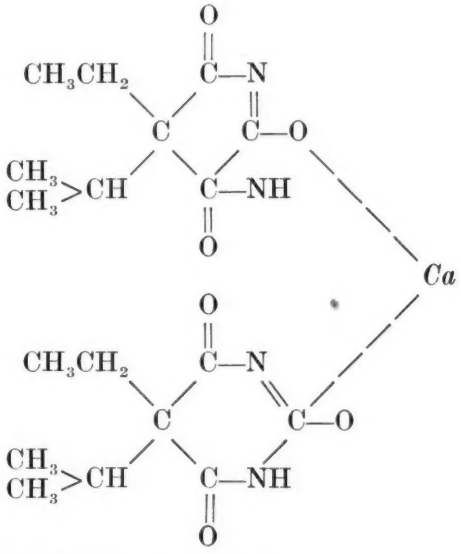
Barbital

Experimental work indicates that dial accumulates in the subcortical regions of the brain but has little influence on the excitability of the motor cortex. The effect of dial on the medullary centers, even after large doses, appears to be comparatively slight unless the drug is injected intravenously at a rapid rate, in which event there is a drop in blood pressure and occasionally, respiratory

difficulty analogous to the intravenous administration of other barbiturates. This drug may be used to excellent advantage as a preoperative sedative in conjunction with general or, more especially, regional anesthesia, in which the malonyl-urea derivatives have been shown to be efficacious in combating the toxic manifestations that may arise from the subcutaneous injection of cocaine or procaine. The average dosage is one and one-half to four and one-half grains.

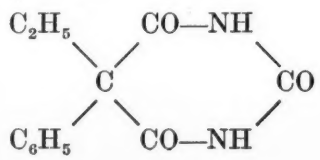
Empirin compound was prepared originally to provide the profession with a commonly prescribed combination of sedative substances. It combines the characteristic analgesic effects of acetyl-salicylic acid, acetphenetidin (phenacetin), and caffeine. This drug has been used a great deal and has been found very helpful in combating the neuralgic pains associated with affections of the dental structure. Its analgesic and sedative effect following exodontia has proved very beneficial. The dosage is one tabloid which contains three and one-half grains of acetylsalicylic acid, two and one-half grains of phenacetin and one-half grain of caffeine.

Ipral is the calcium salt of ethyl-iso-propyl-barbituric acid with the following graphic formula:



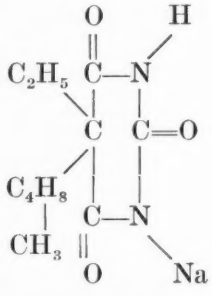
Its action is confined to the central nervous system, especially to the cerebrum, and like other barbituric acid derivatives, it has a sedative action upon the cerebral cortex. The amnesia resulting from the administration of this drug tends to reduce the apprehension and mental distress without interfering with the cooperation of the patient. Dosage is four to six grains.

Luminal is phenyl-ethyl-malonyl-urea, also known as phenobarbital, or phenyl-ethyl-barbituric acid, with the following structural formula:



Many authors consider luminal the most powerful of the true hypnotics, and as such it should be administered cautiously and with great care especially in large doses. Large doses may occasionally be required, but should be given only to patients who are under constant supervision. To produce surgical semi-anesthesia a large dose (fifteen grains by mouth to an average sized patient) may be given all at once about three hours before operation, provided the blood pressure is not low. This will produce a condition of semiconsciousness from which the patient can be awakened. Only small amounts of supplemental anesthesia will be required when this technic has been carried out, and all preliminary mental hazard and worry has been obliterated with the patient at ease.

Nembutal is sodium-ethyl-iso-methyl-butyl-barbiturate, also called pentobarbital sodium, with the following chemical formula:



It is the isomer of amytal, that is, it is composed of the same elements in the same proportion by weight, but differing in one or more properties owing to difference in the chemical structural formula. Yet this small chemical change is sufficient to double the hypnotic efficiency, nembutal being twice as efficient clinically as amytal. In its clinical use, it is the solubility in nerve lipoids and the adsorption upon various colloids which are the important factors. Nembutal has a profound sedative but short hypnotic action; hence a smaller dose is required which yields a correspondingly shorter duration of effect, resulting in a more rapid recovery with practically no excitement or delirium stage. Its use as a preoperative sedative, regardless of the supplemental anesthetic used, is distinctly valuable in that smaller amounts of the additional anesthetic are required and that the period of recovery is shorter. The fact that the short-acting barbiturates are quickly detoxified reduces the incidence of secondary aftereffects, in comparison to the long-acting compounds whose rate of destruction and elimination is slow. Paradoxically, the more toxic members are the safest within therapeutic limits, and especially so when prescribed over a long period of time. Extensive research as to the pharmacology of this drug has shown that it is destroyed in the body, for the most part in the liver. The more rapid induction with nembutal observed clinically may be explained on the basis of the higher distribution coefficient of the ethyl-iso-methyl-barbituric acid which would aid in the rapid transfer of the drug from the stomach or intestine to the nerve and brain centers. The literature contains many tables and charts showing the comparative action of nembutal to the other drugs in regard to efficiency, toxicity, etc., but time and space will not permit to include them in this paper. We do know that it is ideal for preanesthetic sedation, es-

pecially for dental work, where we wish to prevent mental shock and have a relaxed patient who will cooperate. The average dose is one and one-half to three grains, that is, one or two capsules by mouth.

Ortal sodium is the sodium salt of normal hexyl-ethyl-barbituric acid. It is distinctly a hypnotic which acts rather rapidly, inducing a quiet, restful sleep. Clinically the therapeutic dose sufficient to prepare a patient for the administration of an inhalation anesthetic is small in comparison to the anesthetic dose; hence it may be used with safety because it is apparently destroyed in the body in a relatively short period of time, and there is no tendency to accumulation of the drug in the tissues. Because of this comparatively brief action, the effects do not linger any longer than is necessary; hence, no so-called hangover. This drug, therefore, may be classified as one of the quick acting and milder types of barbiturates and is very effective in preoperative sedation prior to the administration of local or general anesthetics. Average dose is three to six grains.

Sodium alurate is one of the most recent drugs that has appeared on the market. It is the sodium salt of allyl-iso-propyl-barbituric acid. It enjoys prompt action, rapid elimination, and has a wide margin of safety. The amount of supplemental anesthetic is appreciably less, and the period of postoperative supervision is lessened somewhat because of the early response, as soon as the effects of the anesthetic begin to disappear, because of the rapid inactivation of the drug. Its potency is about five times that of barbital and more than twice that of luminal; yet it enjoys a wider margin of safety in anesthesia than either of the two. It may be stated that this drug, when properly used, permits the anesthetist to reduce the volume of the ensuing anesthetic, thereby diminishing the untoward aftereffects which are occasionally associated with anesthesia. Large doses of sodium alurate are not necessary when an opiate is to follow in the premedication, because the two synergize and produce the desired effect.

Morphine, pantopon, and the placebo *sodamint* should be mentioned before stating our conclusions. The use of morphine has become so routine that no further comment is necessary. Pantopon, however, is a very valuable drug. It is a highly purified opium preparation containing all the alkaloidal principles of opium in the water-soluble injectable form of hydrochlorides. Pantopon required to produce the requisite analgesia and sedation contains a much smaller amount of morphine than would otherwise be required. Atropine should be used with both morphine and pantopon in the hospitalized cases in order to dry up the secretions and render the field of operation as dry as possible besides making the anesthesia more satisfactory. In certain neurotic and easily excitable people a sodamint or some other similar simple placebo has been known to have produced the desired effect without causing any depression of the brain cells. I mention these simple placebos, given merely to satisfy the patient, which I believe should be used more often in office practice in order that no embarrassment may ensue because of the improper use of promiscuous drugs.

CONCLUSIONS

The drugs I have just described are those most commonly used today and, as such, include not only the milder but also the more potent ones. Although barbiturates as a group possess certain characteristics in common, im-

portant differences do exist. From this list the dentist may select the proper drug for each individual hospitalized case, having in mind the essential clinical knowledge of each particular drug. Most of these drugs have been accepted by the Council on Pharmacy and Chemistry, whose purpose it is to gather and disseminate information on drugs to protect the medical profession in prescribing and using them. Thus has been created an extralegal method of protecting still further the sick, and advancing scientific medicine.

Few patients can contemplate or tolerate the induction period of inhalation or local infiltration anesthesia without anxiety. Despite the fact that its skilled induction according to modern methods is much less unpleasant than formerly, it still remains an ordeal that is dreaded by many. This dread or fear can now be absolutely controlled by the proper and sane use of these modern drugs which we use in our premedication. Finally, remember to use these drugs cautiously and *only* in patients who will be under your direct supervision and for whom you will be legally responsible.

520 Commonwealth Avenue

Department of Orthodontic Abstracts and Reviews

Edited by
DR. EGON NEUSTADT, NEW YORK CITY

All communications concerning further information about abstracted material and the acceptance of articles or books for consideration in this department should be addressed to Dr. Egon Neustadt, 133 East Fifty-Eighth Street, New York City.

Health Dentistry for the Community. A Study of Present Needs and General Trends in the Provision of Community-Wide Dental Care. By the Committee on Community Dental Service of the New York Tuberculosis and Health Association. The Medical Economic Series, Michael M. Davis, Editor. Chicago, 1935, the University of Chicago Press.

The foreword propounds the fact that the study was undertaken to supply the omission of dental conditions in their relationship to dental health of the public and to delineate the rôle of dentistry in the present changing social order. It lays down four primary objectives which are commendable. The subcommittee which conducted the survey included John T. Hanks, Chairman, William J. Gies, John O. McCall, Waldo H. Mork, Harry Strusser, Alfred Walker, Pauline Warner, and Leuman M. Waugh. The Julius Rosenwald Fund is given credit for underwriting part of the cost of publication. Michael M. Davis, Ph.D., is especially mentioned above all others for his constructive criticism, advice, and encouragement.

The introduction, Chapter I, endeavors to define its aim of rendering complete dental care in childhood as a community project. The study is based on observation of three classes in the population, namely, all children, adults who have retained almost all their teeth, and those adults whose mouths have been badly damaged by dental disease necessitating considerable treatment and artificial replacements. It then proceeds to analyze the cost of dental care in the various classes followed by a résumé of previous studies which the compilers feel are based upon "unjustified assumptions." It draws attention to the fact that dental care for children had been overlooked. Care for the preschool child is based on the findings in the Guggenheim Clinic, believing that the money saved by benefiting the health of the school child may be utilized for a universal dental health program for children. By viewing the benefits of the elimination of dental disease as a corrective for systemic disturbances, the need for "community dental care should be regarded as a fundamental health measure."

Chapter II deals with dentistry for children. It includes allusion to systematic dental care by Morrey, diets by Mellanby, McCollum, Hess, Howe, Hanks, Hawkins, Jones, Kugelmass, Bunting, Boyd and Drain, coming to customary conclusions. Quotations, tables and data of various projects over variable periods are cited; among them the Nathan Marks Orphan Home, Mulberry Health District, Guggenheim Clinic, Clara Wachtel Clinic, Oyster Bay Public

School, the St. Louis Survey, the Peoria Program, U. S. Public Health Survey, the New Jersey Civil Works Administration Project, the Floral Park Plan and The Oral Hygiene Committee of Greater New York. The entire condensation is commendable and laudable, being a stimulus for those who seek to know more. Why the Battle Creek Plan with its commendable features was overlooked, is a matter for speculation.

Chapter III deals with adults of various classes showing the cost of care under varying conditions based on figures of the committee on the Cost of Medical Care and T. E. R. A. Clinics. The number of patients to each dentist and the relationship of the amount of work incident to the prevalence of caries in various group ages are delineated. This is a very commendable survey. The cost of the various services as rendered by a self-supporting pay clinic is also analyzed. Incidentally it proves this service could be rendered at the same cost in the office of any practitioner who caters to this type of clientele.

The Industrial Diagnostic Service, instituted by the Chicago Dental Society in view of the good showing of the composite figures, should have received a more cordial reception than the footnote comment on page 40. As a modicum for adult dental health education it has proved useful, but the footnote has turned its favorable aspects into an argument for the need of health insurance.

It further delineates budgeted dental service, cost of dental care in private practice, and touches on restorative dentistry for the lower income group. It advocates universal dental care for children as the only solution, on the premise that prevention is cheaper than cure.

Chapter IV deals with the trend toward socialization. The compilers here set forth all the apparently good arguments for a socialized service. The word "apparently" is used advisedly because advantages are listed without giving cognizance to the disadvantages and the glaring defects of all services of this type.

Chapter V, the "Summary and Conclusion," shows, as does the previous chapters, the guidance and practiced hand of the editor, Michael M. Davis. Although the introductory remarks were disarming regarding the sponsoring of any "plan of so-called socialized dentistry" this chapter advocates the use of the knowledge of existing conditions "to utilize them as a basis for the statement that a comprehensive plan for socialized dental service is urgently needed."

It contains in addition four appendices—first, the Strusser Plan of Universal Dental Care for Children advocated by Harry Strusser, D.D.S., Chief of the Dental Division of the New York City Board of Health; second, the Services and Fees for Dental Care of the State of New York Temporary Emergency Relief Administration; third, the Summary of Dental Conditions as quoted in "Health Dentistry for the Community"; fourth, a bibliography of the Reference Sources.

A well-compiled index which is attached should prove very useful.

It is a very comprehensive little book, well compiled, excellently edited, informative, and a necessary addition to the library of every one interested in the subject.

Leonard Kohn.

Dental Anatomy. A Graphic Presentation of Tooth Forms With an Original Technic for Their Reproduction. By M. Diamond, New York, 1935, Ed. 2, The Macmillan Co.

"The primary objective in the reconstructive endeavor of part or the whole of the dental apparatus is the restoration of maximum function. To that end, a knowledge of the design of the apparatus as established by nature is vital." With this statement, the author acknowledges at the start the emphasis on functional considerations which he carries out in his presentation and which adds a good deal of lively interest to his textbook.

After a discussion of tooth development and tooth tissues (which unfortunately lacks histologic data and references) the anatomic landmarks of the teeth

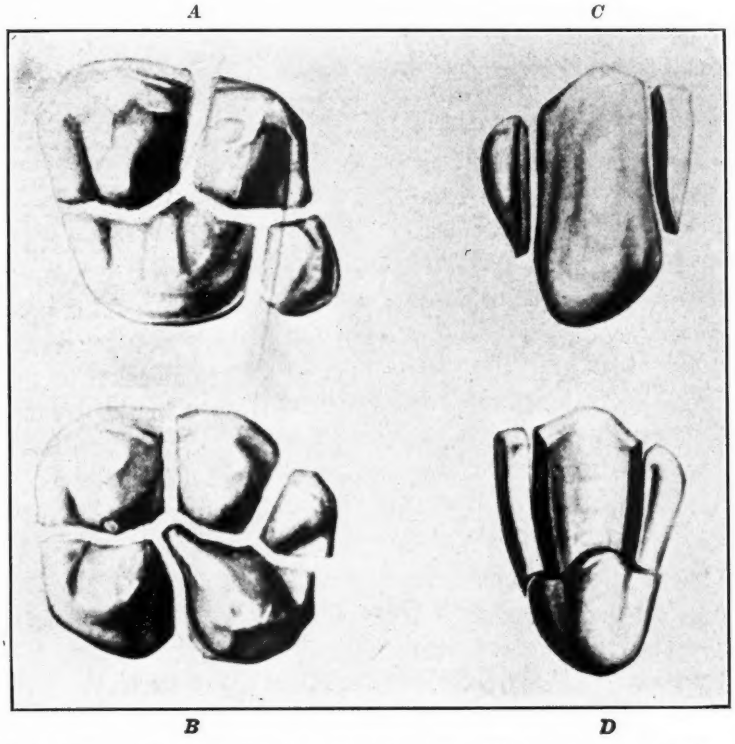


Fig. 1.—Diagrammatic representation showing proportions of: *A*, maxillary first molar; *B*, mandibular first molar; *C*, labial aspect of maxillary cuspid, showing proportions of three labial lobes; *D*, lingual aspect, showing proportions of lingual lobe and the three labial lobes.

are described. These landmarks should serve as guideposts for an easier understanding of anatomic details and for reproduction of crown forms. They are also related to functional requirements.

The most important landmarks and those which form the basis of all anatomic description and reproduction technic are derived from a division of each tooth crown into developmental lobes. Practically all crowns are formed from four lobes. In the incisors and canines, three lobes make up the labial half, and the fourth, the lingual (Fig. 1 *C* and *D*). In the bicuspids, three lobes form the labial and one the lingual part, with the exception of the mandibular second bicuspid, in which two lingual lobes are present. Of the four lobes comprising the maxillary molar crowns, two are buccal and two lingual (Fig. 1 *A*), but in

the three-lobe maxillary third molar, the fourth lobe disappears from the lingual half. The crown of the mandibular first molar has three buccal and two lingual lobes (Fig. 1 *B*), the rest of the mandibular molars have four lobes.

The technic of tooth reproduction is taught by means of the "symmetrical tooth form." This latter constitutes the "basic form of the crown of the tooth stripped of variations and anomalies." Mechanical drawings illustrate each step of this original method of reproduction, and they are aided by measurement tables.

The chapter on the arrangement of teeth and occlusion deals with the arch form of maxilla and mandible, the inclination of tooth crowns and roots, the occlusal plane, and the curve of Spee. Here we also find a résumé of the differentiation between tooth eruption and tooth elevation (originally defined by the reviewer in the *Journal of Dental Research* vol. 12, but somewhat misinterpreted by Diamond). Tooth eruption is characterized by a recession of the gingival tissues, while tooth elevation signifies the occlusal growth of a tooth together with its investing tissues whenever occlusal contact is lost.

The tissues surrounding and supporting the teeth include: the peridental membrane, the alveolar bone, and the gingival tissues. Pulp chamber and pulp canals are described in detail, and the outline of the canals is demonstrated by macroscopic sections through the teeth in different directions and by a series of radiographic pictures. The description of the deciduous teeth contains data regarding the development of the alveolar process and the resorption of the deciduous teeth, besides tables of calcification time and tooth eruption.

One of the most valuable chapters is the one dealing with variations and anomalies. The variations are divided into natural variations (congenitally predetermined), brought about by changes in the development of the individual lobes, and in acquired variations, which are due to metabolic disturbances. (Functional modifications like attrition or erosion would fit more appropriately in the chapter on occlusion, the result of which they are.) Dental anomalies are classified into supernumerary teeth, gummas or twinned teeth, congenital absence of teeth, and unusual forms, affecting either the crowns or the roots. The rest of the book is devoted to the muscles of mastication, the blood and nerve supply, the temporomandibular articulation, and the oral cavity with its soft tissues.

The author's original method of dividing each tooth into individual lobes adds a great deal to the easier understanding of tooth form. Although this division is not substantiated by embryologic development or histologic calcification centers, it makes an excellent working theory. Together with the "symmetrical" tooth form it offers, no doubt, the best available teaching method for the reproduction of tooth forms by dental students. The nomenclature, always a vulnerable part of textbooks on anatomy, has been carefully and logically prepared throughout the volume, and is further aided by a comprehensive glossary. The bibliography, on the other hand, is sadly neglected (containing only twelve names) and does not present more than a fraction of the material used in the volume. It might be well to suggest additional reading matter for the interested student. The illustrations are abundant and excellent, and so are the print, paper, cover, and arrangement of the text.

E. N.

The Forum

Articles for this department should be sent to Dr. William R. Humphrey, 1232 Republic Bldg., Denver, Colo.

More About Treatment of the Deciduous Dentition

The question, "What stage in the development of dentition is best suited for initiating orthodontic treatment?" gives the impression that all cases of malocclusion are identical. If this were true, it might be possible to formulate a fixed rule to cover the correct time for treatment, together with a plan of treatment which could be followed by any mechanic. Each case, however, is an individual problem, and a decision as to the best time for treatment can be arrived at only after a careful consideration of many possible factors.

The statement that "there is no justification for the treatment of malocclusion, of whatever form, in the deciduous dentures" is entirely unwarranted and disregards the results of orthodontists who have treated deciduous dentures with gratifying success. Mention has been made of treating children of "five or six years of age" with no benefit to the patients. At this stage of development considerable absorption of the roots of the deciduous incisors is usually shown by the x-ray films, and consequently the movement of these teeth is contraindicated.

I cannot endorse the statement that the best time generally to commence corrective orthodontic treatment is while the deciduous molars are being shed and replaced by premolars. It might be the best time in some cases. In other cases, it is a good time to permit the permanent first molars to move forward naturally, without orthodontic interference, and to produce proximal contact of the premolars.

Charles R. Baker.

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Editorial

To the Members of the American Society of Orthodontists

A great man has truly said: "The world belongs to the energetic." The progress of our profession and of the American Dental Association has resulted from energy being capably employed in experimentation and the development of new ideas.

Because our record shows steady and uniform progress, we cannot assume that it will continue unless we make earnest effort. We must be openminded to original ideas and must foster independent research. We must encourage experimentation while realizing that every new technic will not be workable. We must be sympathetic to new ideas while realizing that the value of many will be but remote.

Our organization is a means of uniting individual effort, and union is necessary if we are to profit and progress. All men do not think along the same lines. Original thinking may lead to important discoveries. New ideas, put in the crucible of constructive criticism, sometimes prove to be of great value. Thoughtful, resourceful men may originate something which enables them and the profession to make important advancement.

We must not be content with the ever faithful—with the veterans who have borne the heat and burden of the day. While recognizing the importance of their service, we must broaden our vision to include those joining the ranks, welcoming them gladly so that our numbers may ever increase.

In view of the great effort being made to anticipate, direct, or check legislative action, and the many schemes—altruistic, socialistic, or cooperative—being bruited about, it is important that we have a corps of willing workers. To render effective service, they must be trained, officered, and advised by seasoned, capable, and experienced leaders.

This is no time for lethargy. Programs must be developed for local organizations that will retain or revive interest. A multitude of new ideas is in the air. Some endanger our future. There is much to give thought to, much to discuss, much that is new to welcome in part or in whole, and much to avoid or reject.

We must work without ceasing to maintain our *esprit de corps*. Men of originality are needed for scientific research. However, for the American Dental Association to continue to function and serve the profession to the utmost, we need many plain, everyday working men. They are the foundation of our organization, which numbers forty thousand. The American Dental Association has an intricate network of branches, functioning with machinelike rhythm. Its welfare and betterment depend upon new individuals and new ideas being incorporated into it daily.

Many who are now outside the organization belong within it. It is important to the profession for them to join the American Dental Association, and it will be the means of their gaining contact with the latest thought and the results of the latest experimentation. Adding them to our group will help to assure us an active, vigorous, vibrant organization. For all to profit, we must have the cooperation of all. Each officer and member must do his utmost to increase our enrollment. We must not rest until every ethical dentist is an active member of local, state, and national organizations.

GEORGE B. WINTER, President
American Dental Association.

News and Notes

American Society for the Promotion of Dentistry for Children

The annual meeting of the American Society for the Promotion of Dentistry for Children will be held in the Francis Hotel, San Francisco, Calif., on July 13 and 14, 1936. Interesting and helpful papers, table clinics, and practical demonstrations will be given covering all phases of dentistry for children. A round table luncheon will be held on Monday, July 13, at 12:30. All sessions will be held at the Francis Hotel, and every member of the American Dental Association and the Canadian Dental Association will be most welcome.

WALTER T. MCFALL, President.
JOHN C. BRAUER, Secretary.

American Dental Assistants Association

The twelfth annual meeting of the American Dental Assistants Association will be held in San Francisco, Calif., July 13 to 17, 1936. Headquarters will be at the Hotel Whitcomb. For further information please address

LUCILE S. HODGE, General Secretary,
401 Medical Arts Building,
Knoxville, Tenn.

British Empire Dental Meeting

The British Empire Dental Meeting will be held in London during the last week of July. A large delegation of Canadian dentists will be in attendance and will leave Montreal on July 10. Dr. Fred J. Conboy of 86 Bloor St. W., Toronto, Ontario, is chairman of the Arrangements Committee.

Postgraduate Course in Periodontia at New York University College of Dentistry

The New York University College of Dentistry will hold its third annual postgraduate course in periodontia for two weeks, full day; or for four weeks, mornings only, beginning July 6, 1936. Courses are limited to fifteen men.

The course will include etiology, diagnosis, and treatment of periodontal disease. Various technics of pocket eradication will be considered and conservative treatment stressed. Vin-

cent's infection; diagnosis of types of bone resorption; mouth manifestations of systemic disease; periodontal foci of infection; toothbrushing; instrumentation; balancing of occlusion. Taught by lectures and clinical work, each student treating several cases.

Instruction by Drs. Samuel Charles Miller, Sidney Sorrin, J. Lewis Blass, and the entire periodontia faculty.

For information and application, address Periodontia Department, New York University College of Dentistry, 209 East 23rd Street, New York, N. Y.

SAMUEL CHARLES MILLER,
Associate Professor of Periodontia.

Ninth International Dental Congress of the F. D. I. in Vienna

In Vienna, Austria, from August 2-8 inclusive, the Ninth International Dental Congress of the Federation Dentaire Internationale will be held.

American Association of Women Dentists

The American Association of Women Dentists will meet at San Francisco, July 13 to 17.

DR. VIRGINIA TREMBLY,
317 Burns Building,
Colorado Springs, Colo.

The 78th Annual Meeting of the American Dental Association

The Annual Meeting will be held at San Francisco, California, July 13 to 17, inclusive. This meeting is most important, and the time and the place are ideal for a vacation. An occasion will be afforded to meet friends of other days and other places. A chance will be given to hear the ablest and most far-seeing of the profession. There will be plenty of opportunity to see the newest appliances, to study the latest theories, and to make contacts, new and old.

One cannot stress too much the meetings of the scientific sections and the clinics. They have great professional interest and value. There are outstanding men on the program, and real contributions will be made that will be worth traveling over a continent to listen to. If one would keep up to date professionally, attendance is well-nigh imperative.

Apart from the health and pleasure and recreation, and aside from the immense value of contacts and the freshening and revivifying and modernizing of the varied skills and scientific knowledge, there is another phase of the meeting that is trenchant.

Change in all things is inevitable. Some changes are perceptible; some, imperceptible. Some have a passing effect; some, a profound one. The changes witnessed by this organization since the last annual meeting have been of extreme importance. These changes affect all of us, consciously or unconsciously. They are of all kinds—economic, social, legislative, bureaucratic, and what not. Some of them we may like; some we may choose; some may be forced upon us. But whether we like, or whether we choose, or whether we are to have certain things forced upon us, it is certainly well and wise to be cognizant of the situation with which we are face to face. I know of no way to become better acquainted with what the future holds in store for us than to be present, if it is at all possible, at this Annual Meeting, where we shall have the best thoughts of the best minds of our profession.

A great prophet once asked, "Can the Ethiopian change his skin or the leopard his spots?" One might ask now, "How much can American dentistry change from its ideas and ideals of the past?" We shall probably be hard-put-to-it not to give up some of what has been called our rugged individualism.

There is little to be gained in mourning over the inevitability of change. Shall we continue to be stand-patters? Is it wiser to inaugurate some changes ourselves? These are some of the problems that will meet us when we meet at the Golden Gate. May the spirit of prophecy descend upon us and may dentistry give fitting answer to the queries that confront us with gathering urgency.

Be sure to enter on your calendar, "July 13 to 17, San Francisco, California."

GEORGE B. WINTER,
President, American Dental Association.

Omicron Kappa Upsilon

The members of Omicron Kappa Upsilon will meet for a good fellowship luncheon at the St. Francis Hotel, San Francisco, during the meeting of the American Dental Association to be held in that city July 13-17, 1936. Announcement as to the date and hour will be made in a later issue of this Journal.

Dr. Fred T. West of 2595 Mission Street, San Francisco, is chairman of the local committee in charge of the luncheon.

ABRAM HOFFMAN,
Supreme Secretary-Treasurer,
311 East Chicago Avenue,
Chicago, Illinois.

Note of Interest

Dr. Norman L. Hillyer announces the removal of his office to 1 East 57th Street, New York, N. Y. Hempstead office—Professional Building, N. Y. Practice limited to orthodontia.

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